EXHIBIT A

EXHIBIT 117

American Journal of Industrial Medicine 3:259-311 (1982)

Occupational Exposure to Asbestos: Population at Risk and Projected Mortality—1980-2030

William J. Nicholson, PhD, George Perkel, MA, and Irving J. Selikoff, MD

Estimates have been made of the numbers of cancers that are projected to result from past exposures to asbestos in a number of occupations and industries. From 1940 through 1979, 27,500,000 individuals had potential asbestos exposure at work. Of these, 18,800,000 had exposure in excess of that equivalent to two months employment in primary manufacturing or as an insulator (≥ 2-3 f-yr/ml). 21,000,000 of the 27,500,000 and 14,100,000 of the 18,800,000 are estimated to have been alive on January 1, 1980.

It is further estimated that approximately 8,200 asbestos-related cancer deaths are now occurring annually. This will rise to about 9,700 annually by the year 2000. Thereafter, the mortality rate from past exposure will decrease, but still remain substantial for another three decades.

Key words: asbestos, occupational exposure, risk assessment, morality projections

INTRODUCTION

A large volume of research has been conducted on the adverse health effects of exposure to asbestos. However, relatively little is known about the magnitude of the population at risk to asbestos-related disease. A number of occupations and industries have been identified as involving substantial occupational exposure to asbestos, but no detailed evaluation has been made to quantify the number of persons whose employment experience has resulted in sufficient exposure to warrant characterizing them as at risk. This analysis is designed to provide an assessment of the extent and consequences of occupational asbestos exposure in the United States between 1940 and 1979.

The task of estimating the population at risk to asbestos-related disease is complicated by a number of factors:

Environmental Sciences Laboratory, Mount Sinai School of Medicine (CUNY), New York.
The analysis was prepared as part of a study for the US Department of Labor entitled "Disability Compensation for Asbestos-Associated Disease in the United States," June 1982.
Address reprint requests to Dr. William J. Nicholson, Environmental Sciences Laboratory, Mount Sinai School of Medicine (CUNY), One Gustave Levy Place, New York, NY 10029.
Accepted for publication July 23, 1982

0271-3586/82/0303-0259$14.00

LC 000738

1. The precise number of persons occupationally exposed to asbestos at any given time is not known.

2. The level of exposure to asbestos necessary to increase the risk of incurring asbestos-related disease is only imperfectly known, estimates being complicated by the varying interactions of the two elements that go into "dose" (time and intensity).

3. The extent to which workers have changed occupations and/or industries from time to time so as to place them at risk to asbestos-related diseases (or to end such exposure) at any time in the past four decades is not known.

We have sought to overcome these obstacles by compiling the best available data concerning worker exposure to asbestos and the turnover of workers in the occupations and industries involved. The sources and methods used to estimate the population at risk are set forth below.

MATERIALS AND METHODS

Identification of Industries and Occupations at Risk

Workers are exposed to asbestos in a wide variety of industrial pursuits from mining and milling to primary manufacturing (producing manufactured goods from raw asbestos fibers) to secondary manufacturing (processing asbestos manufactured products to make other products) to consumer industries (utilizing a finished product containing asbestos without modification) [Daly et al, 1976].

Mining and milling. Fewer than 600 persons in the United States are employed in mining and milling asbestos [Meylan, 1978]. In view of the small number involved and the lack of information on employee turnover, we have excluded this industry from our estimates.

Primary manufacturing. The Asbestos Information Association has estimated that there are upwards of 3,000 discrete uses of asbestos. A selection of major asbestos products and their uses is presented in Table I. The primary manufacturing industries in which asbestos products are produced and which involve substantial asbestos exposure to production and maintenance employees are as follows:

Asbestos products industry (SIC 3292). The major products of this industry are friction products, asbestos-cement pipe and sheet, asbestos textiles, floor tiles, roofing felts, insulating materials, and other asbestos building materials.

Extensive data indicate that excessive fiber concentrations existed in the production of asbestos products during previous years. In a study of retirees from one of the largest asbestos products manufacturers, Henderson and Enterline [1979] categorized work exposures according to total dust concentration (as measured by a midget impinger) times period of employment. Using recently obtained data on the conversion between such particle counts and fiber concentrations, it is estimated that the average concentration to which the members of his cohort were exposed was 30 fibers/ml [Asbestos Information Association, 1979]. Similar concentrations were suggested for the work force exposure in a large United States asbestos products manufacturer studied by Nicholson et al [in press]. Here subjective data, consistent with company measurements of dust concentrations, suggested that the person-weighted average exposure was approximately 25 fibers/ml between 1945 and 1965. In two asbestos insulation manufacturing facilities in Port Allegany, Pennsylvania, and Tyler, Texas, aver-

A study workers exposed to brominated chemicals in three plants provides data on the distribution of employment times of all 3,579 individuals employed in the facilities [Wong, 1981]. It substantiates the presence of a large number of individuals with very short employment times. Of all employees, 16.4% worked for less than one month and an additional 28.5% for 1-5.9 months. The full distribution of employment times can be characterized by a two-component decreasing exponential. Thus, the work force can be considered as made up of two groups. The average employment time of one, consisting of approximately 2,200 individuals, was 0.5 years and of the other, with 1,400 individuals, was 11.7 years in good agreement with the data of Table XIII.

Relative Risk by Industry

To calculate the asbestos-related cancer mortality in a given industry or operation, it is necessary to have an absolute or relative measure of exposure for the employee group. While detailed information is not available on the asbestos air concentrations that have been prevalent in previous years in each of the above industries, estimates can be made of the relative risk of death from asbestos exposure on the basis of a variety of other studies. In the calculation of asbestos-related cancer mortality for a given industry or occupation, we will utilize the available data for insulation workers for the dose and time dependence of asbestos cancer. To translate available data for insulation workers to other industries, it is necessary to establish measures of exposure for the different groups considered at risk relative to that of insulation workers. These *relative risks for equal times of employment* will be determined by three indices. The primary one is the directly measured mortality data, especially that of mesothelioma or lung cancer, in an industry or trade. A second is the directly measured average concentrations of asbestos that can be attributed to the work activity. The third is the prevalence of X-ray abnormalities after long-term employment in an industry. Here, we will assume that the percentage of X-ray abnormalities attributable to an exposure circumstance after 20 years of employment will be proportional to the total dose of asbestos inhaled by the workers in that industry. Where the percentage of abnormal X-rays approaches 100%, the relative risks will be determined using the percentages of X-rays having a category 2 or greater abnormality on the ILO U/C scale. Information on these direct and indirect measures is shown in Table XV along with the sources of the various data.

For industries in which none of the above indices are available (construction, railroad steam engine repair) or for which the data are very uncertain, relative risk estimates were made from the numbers of mesotheliomas identified among individuals in different asbestos exposure circumstances compared with the total work force exposed. These data utilized the nationwide survey of mesothelioma in 1972 and 1973 by McDonald and McDonald [1980]. The numbers from this series are shown in Table XVI.

The relative risks, by industry, estimated from all of the above data, are listed in Table XVII. Also indicated in Table XVII are the principal data sources considered in the relative risk estimates. The data available for the estimates are limited and the estimates are necessarily approximate. For the years 1972-1979, the relative risks for manufacturing, insulation work, shipbuilding, and utility employment will be reduced to 0.1, and those of the other industries (except automobile maintenance) to 0.05 to reflect the adoption of control measures. Further, exposures subsequent to 1979 will

TABLE XV. Indices of Relative Asbestos Exposure in Selected Occupations and Industries

Industry of occupation	Estimated average fiber concentrations	Relative risk of lung cancer	Percentage of deaths from mesothelioma	Applicable employment period (years)	Percentage of: parenchymal abnormalities 1+	parenchymal abnormalities 2+	pleural abnormalities	Applicable employment period (years)
Primary manufacturing	20–40	2.8[a]–6.1[a]	2.6[a]–9.1[a]	1–20+				
Insulation work	15[c]	4.8[c]	8.7	20+	85[c]	42[c]	56[c]	20+
Shipbuilding and repair	2[c]	1.8[c]		2–3	85[c]	17[c]	54[c]	20+
Chemical plant and refinery maintenance		1.5[c]		15 or	33[c]	[c]	44[c]	20+
Automotive maintenance	0.1–0.3[c]				[c]			10+
Marine engine room personnel							16–20[c]	15

[a]Nicholson, 1981a.]
[b]Seidman et al. 1979.]
[c]Selikoff et al. 1979.]
[d]Selikoff et al. 1965.]
[e]Selikoff, 1965.]
[f]J. Thomson, quoted in Enterline, 1981.]
[g]Lilis et al. 1981.]
[h]Selikoff et al. 1981.]
[i]Hans et al. 1979.]
[j]Lilis et al. 1980.]
[k]Nicholson, 1982.]
[l]R.N. Jones, 1980.]

EXHIBIT 118

1 of 1 DOCUMENT

Guide to Toxic Torts

CHAPTER 10 Proof of Causation *

2-10 Toxic Torts Guide § 10.01

§ 10.01 Causation Constitutes the Primary Problem in Toxic Tort Litigation

[1] Overview of Medical Causation

In a toxic tort action, in addition to proving product defect causation, a plaintiff must prove "medical causation," i.e., that the plaintiff's injuries were proximately caused by exposure to the defendant's products.n1 Proof is is more subtle and sophisticated in the toxic tort context than in more traditional torts.n2 The problem of proving medical causation with respect to a specific defendant's products is compounded where a plaintiff has been exposed to multiple products of multiple defendants over an extended period of time.n3

There is almost universal agreement among commentators that proving causation is the most significant problem in toxic tort litigation. The issue has been the subject of extensive legal writing.n3.1 It has been one of the major concerns of an extensive governmental study,n4 and has been a grave concern not only of the legal system but the scientific community as well.n4.1

Yet, according to some courts and commentators in the early 21st century, the modern trend in toxic torts cases is to relax or broaden the standard of determining medical causation. This trend is due to recognition that establishing a nexus between a defendant's conduct or product and a plaintiff's injuries in toxic torts cases is more subtle and sophisticated than proof in traditional tort cases. A less traditional standard is essential because of the issues mentioned above, i.e., long-term exposures to multiple toxins, and harm resulting from biochemical disruption or acute toxic substance.n4.2

This chapter analyzes the problems in proving causation in toxic tort litigation. It discusses the available approaches to proving causation and suggests some developing alternatives.

[2] Definitions of Causation

[a] "Substantial Factor" Test and "But For" Test. Causation refers to the factual and legal relationships that must be established between tortious conduct and injury before our legal system will impose liability. In order to establish negligence there must be a legal duty to use care, a breach of that duty, and proximate cause between the breach and the injury.n4.3 In a general sense, the problem of causation raises a variety of issues, such as causation in fact, proximate cause, and apportionment of damages.n5 The principal focus of this chapter is the issue of causation in fact.n6 The traditional approach to causation in fact was the "but for" or "*sine qua non* " test. For example, in an early case in which a plaintiff was injured when he fell on a stairway it was alleged that the failure of the defendant to provide a handrail caused the plaintiff to be injured. In reversing a verdict awarding the plaintiff damages, the court held that the absence of a handrail was not the cause of the plaintiff's injuries.n7 Under this test, causation in fact was established when it was proven that the injury would not have occurred "but for" the defendants' tortious conduct. As the late Dean Prosser argued, the rule was more a rule of exclusion than a definition of causation.n8 If the injury would have occurred in any event, then it was thought that the tortious conduct was not the cause in fact. Unfortunately, this simple rule did not work in all circumstances, particularly when two separate potential causes combined to create a loss.

The limitations of the traditional "but for" test gave rise to a new approach, the "substantial factor" test. Under this approach, a potential cause is deemed to be the cause in fact if it was a substantial factor in bringing about the result.n9

The substantial factor test for causation rather than the "but for" test should be applied in a case involving multiple causes of a particular harm. Under the substantial factor test, a cause must be sufficient before it can be substantial; the critical question is whether the defendant's toxic substances or conduct were independently sufficient causes of harm to the plaintiffs.n9.1 The first case in which a court applied the substantial factor test was *Anderson v. Minneapolis St. Paul & Ste. Marie Railway Co.,* n10 in which the court stated that the defendant's conduct is a cause of an event if it is a material element and a substantial factor in bringing it about.

The substantial factor test is satisfied by proving that it is more likely than not that the conduct of the defendant was a substantial factor in bringing about the harm.n11 The substantial factor has been applied in toxic tort cases, and the courts have ruled on what constitutes sufficient proof that the defendant's conduct was substantial factor. For example, in a recent New York asbestos case, *Diel v. Flintkote Co.,* n12 the court held that in New York, plaintiffs must establish that the decedent was exposed to the defendant's product and that it was more likely than not that this exposure was a substantial factor in his injury. In *Sheffield v. Owens-Corning Fiberglas,*n13 the Supreme Court of Alabama stated that admiralty courts often look to the Restatement (Second) of Torts section 431 for the substantive standards of proof of causation. Section 431 provides that an actor's negligent conduct is a legal cause of harm to another if his conduct is a substantial factor in bringing about the harm. In order to prevail, the plaintiff must make it appear that it is more likely than not that the conduct of the defendant was a substantial factor in bringing about the harm.

In determining whether a defendant's conduct was a substantial factor in an asbestos case, the court in Maryland applied a two-step analysis under which "one may reasonably conclude that it is more likely than not that the conduct of the defendant was a substantial factor in bringing about the condition that is the subject of the complaint" only if there is sufficient evidence that the defendant's product was used at the site of the plaintiffs' exposure, and that the plaintiffs worked within the vicinity of the asbestos workers using the product and therefore in the vicinity of the product itself.n14 The plaintiffs in that case testified that they had seen the defendants' names printed on asbestos products at their work place and that they had come into physical contact with asbestos-containing products in the course of their employment. The court found the plaintiffs' evidence to be sufficient, and it rejected the defendants' argument that their activities were not a substantial factor in causing the plaintiff's injuries. Therefore, the verdict in favor of the plaintiffs was affirmed.

A substantial factor has been referred to as a "producing cause"n15 as well as a "major contributing factor."n16 It need not be the only cause. For example, in an asbestos case, *Blair v. Eagle Picher Indus., Inc.,* n17 the 10th Circuit, applying Oklahoma law, upheld the District Court's instruction to the jury as follows:

> You are instructed that a "direct cause" of an injury is that cause which in the natural sequence contributed to producing the injury. It need not be the only or the last cause. It is sufficient if it concurs with some other cause which in combination with it, brings about or contributes to the injury You are further instructed that evidence that a defendant's asbestos containing product was at the ... plant at the same time as [the plaintiff] is not sufficient evidence from which you may find that plaintiff's injury, if any, was directly caused by the asbestos containing product.

Similarly, in a class action asbestos case, *Cimino v. Raymark Indus. Inc.*,n18 the District Court for the Eastern District of Texas held that under Texas law a plaintiff must prove that a defendant's unreasonably dangerous product was a producing cause of a plaintiff's injury or disease. A producing cause was defined as an efficient, exciting, or contributing cause which in a natural sequence of events produces an injury or disease. The court further stated that a plaintiff in an asbestos suit need not show that exposure to a defendant's product was the sole cause of the asbestos-related injury or disease. If exposure to the defendant's product combined with other causes to produce a single injury, the plaintiff may recover from the defendant for the entire injury. The plaintiff need only show that the exposure to a defendant's product was a substantial factor in causing the plaintiff's injury.

In a New Jersey case, *Theer v. Phillip Carey Co.,* n19 the supreme court discussed that, in asbestos failure-to-warn cases, a plaintiff must normally prove two separate elements of causation. The plaintiff must demonstrate that his or her injuries were proximately caused by exposure to defendant's asbestos product, which is known as "medical causation." The plaintiff must also demonstrate so-called product-defect causation, that the defect in the product was a proximate

cause of the injury. Because the alleged product defect was the failure to provide warnings, the plaintiff is required to prove that the absence of a warning was a proximate cause of his or her harm. The court also pointed out that the heeding presumption in failure-to-warn cases serves to ease an injured plaintiff's burden of proof. That objective is especially important because establishing that the absence of a warning was a substantial factor in the harm alleged to have resulted from exposure to the product itself is particularly difficult. The heeding presumption serves to eliminate conjecture about whether a given plaintiff would have heeded a hypothetical warning, and discourages determinations of causation that are based on extraneous, speculative considerations and unreliable or self-serving evidence.

While it need not be the only cause, a defendant's conduct must somehow be responsible for the plaintiff's injuries. Absent sufficient evidence from which a "jury [could] reasonably find that the defendant's conduct was a cause of the plaintiff's harm or injury" the "Illinois courts have, as a matter of law, refused to allow a plaintiff to take the causation question to the jury."n20

Other courts have also held that the question of causation is a matter of law where the plaintiffs were unable to establish that the defendant was in any way responsible for the plaintiff's injuries. In a asbestos action, *Benshoof v. National Gypsum Co.,* n21 the court granted summary judgment in favor of the defendant because the plaintiffs did not present direct or circumstantial evidence of at least one exposure to the defendant's products. The court stated: "Summary disposition on the issue of proximate cause is appropriate when plaintiff's evidence does not establish a causal connection, leaving causation to the jury's speculation' (citations omitted)." In another asbestos case, *Robertson v. Allied Signal, Inc.*,n22 the Third Circuit stated: "Where the relevant facts show either that the defendant was not responsible for the injury, or that the causal connection between the defendant's negligence and the plaintiff's injury is remote the question of causation is decided by the court as a matter of law." The "substantial factor test" retains "but for" causation as an essential precondition. As the court stated in *Robertson v. Allied Signal, Inc.* :n23

> Proof of causation involves two elements: proof of cause in fact and proximate cause. Case in fact or "but for" causation requires proof that the harmful result would not have come about but for the conduct of the defendant. Proximate cause, in addition, requires proof that the defendant's conduct was a substantial contributing factor in bringing about the alleged harm.

In situations in which there are two or more active forces operating in concert to produce the injury, each cause need not be the sole cause. The question is whether each contributing cause alone is a substantial factor in causing the injury. For example, in *Tragarz v. Keene Corp.,* n24 the defendants contended that the district court committed reversible error in refusing to admit evidence of the decedent's exposure to other asbestos products for purposes of demonstrating cause in fact. The Seventh Circuit rejected this contention, explaining that the "substantial factor test" is not a comparative test in which the jury assesses all contributing causes and determines which ones are substantial. Rather, courts are concerned with whether each contributing cause, standing alone, is a substantial factor in causing the alleged injury.n25

The courts in Maryland have held that in order to recover against a particular defendant, the plaintiff must show that the specific defendant's conduct was a substantial factor in bringing about the harm. For example, in an asbestos action, *Owens-Illinois v. Eagle Picher Indus., Inc.,* n26 the trial court instructed the jury:

> In order for a plaintiff to recover against a particular defendant ... [it] must be shown that a product or product manufactured by that defendant, by a particular defendant was a substantial factor in causing the asbestosis.
>
> If no product manufactured or supplied by a particular defendant was a substantial factor in causing the asbestosis, then that defendant has no responsibility. ...

The appellate court held that the instruction fairly and accurately conveyed the term "substantial factor," and that the trial court did not err in refusing to grant the defendant's proposed instructions containing an "expanded" explanation of the term.n27

However, in an earlier case in *Maryland, Gist v. Raymark Indus.*,n28 a jury verdict of $600,000 was overturned because the judge did not believe that the jury understood the charge regarding substantial factor. The defendant claimed its product produced only a small amount of dust and that the type of asbestos used did not cause mesothelioma.

To establish that the product manufactured by a particular defendant was a substantial factor in causing an injury, it has been held that a plaintiff is not required to prove that "but for" the particular product of each defendant he would not have sustained an injury. The plaintiff "need only show that it is more likely than not that the conduct of the defendant was a substantial factor in bringing about the harm."n29 In this action to recover damages for an injury resulting from exposure to asbestos products, the court affirmed the verdict in favor of the plaintiff.

Some jurisdictions still adhere to the "but for" requirement. In an asbestos action, *Money v. Manville Corp. Asbestos Disease Comp. Trust Fund*,n30 the Supreme Court of Delaware stated that, "under Delaware law, establishing proximate cause requires a plaintiff to prove but for the tortious conduct of the defendant, the injury which the plaintiff has suffered would not have occurred." The court went on to state that Delaware law law also requires that the "but for" connection between exposure and injury must be established by expert testimony.n31 Utah requires that a defendant's negligence be a substantial factor and the actual cause of the plaintiff's injury.n32 Florida adheres to the "but for" test in wrongful death cases.n33

[b] General Causation and Specific Causation. Causation in toxic tort cases is discussed in terms of general and specific causation. General causation is whether a substance is capable of causing a particular injury or condition in the general population,n33.1 whereas specific causation is whether a substance caused a particular individual's injury. To establish their claims, plaintiffs must show both general and specific causation.n33.2

General causation addresses whether a substance increases the risk of disease in a group. There is authority that careful scrutiny is required when an expert attempts to demonstrate general causation through analogy to a different but similar substances. This is because even minor deviations in chemical structure can radically change a particular substance's properties and propensities.n33.3

The Kansas Supreme Court had the opportunity to address the need for separate evidence of general causation to admit testimony of specific causation. It noted that courts typically have applied general causation requirements (requiring plaintiffs to present confirming epidemiological evidence) in cases involving mass exposure, with large existing epidemiological records. Conversely, courts have not imposed general causation requirements in cases involving injuries in the "sporadic accident model" of tort cases, in which only a single plaintiff or a few plaintiffs suffered injury due to some exposure.n33.3

To establish specific causation, plaintiff must not only introduce sufficient epidemiological evidence (*see* §§ 10.02[6][c], 10.03[4], *below*), but must also show that he or she is similar to those in the studies. This includes proof that the injured person was exposed to the same substance, that the exposure or dose levels were comparable to or greater than those in the studies, that the exposure occurred before the onset of injury, and that the timing of the onset was consistent with that experienced by those in the study.n33.3.1 In other words, a plaintiff in a toxic tort case must prove the levels of exposure that are hazardous to human beings in general as well as the plaintiff's actual level of exposure to the defendant's toxic substance (*see* § 10.02[1], *below*).n33.3.2

[c] Causation under the Federal Employer's Liability Act (FELA) and the Jones Act.

Railroad workers and seamen seeking recovery from asbestos and other chemicals under federal statutes have an advantage when it comes to establishing causation over the typical toxic tort plaintiff in state court.

The Federal Employer's Liability Act (FELA)n33.4 has a relaxed standard of proof regarding causation. A plaintiff must offer "more than a scintilla of evidence in order to create a jury question on the issue of employer liability, but not much more." The test is whether the proofs justify with reason the conclusion that employer negligence played any part, even the slightest, in producing the injury.n33.5

Although the quantum of evidence necessary to satisfy causation under FELA is relaxed, plaintiffs must nevertheless present some reliable evidence on the issue. FELA does not make the employer the insurer of the safety of its employees while they are on duty, and the basis of its liability is its negligence, not the fact that injuries occur.n35.5.1 In toxic

tort cases, this generally requires reliable expert testimony.n33.6 Courts have not applied FELA's lower burden of proof to the admissibility of expert testimony. The *Daubert* standard of admissibility of expert evidence extends to each step in an expert's analysis in a FELA case.n33.7

In *Gautreaux v. Scurlock Marine,* n33.8 the Fifth Circuit clarified a plaintiff's standard of causation for damages under the Jones Act,n33.9 which provides a right of recovery against a seaman's employer for negligence resulting in injury or death. There is a reduced, "slight," or "featherweight" standard of causation between the employer's negligence and the employee's injuries. The Second Circuit has held that expert testimony on the issue of causation is necessary in Jones Act cases where a lay juror could not be expected to intuit the causal relationship between the acts in question and the injury, and that such expert testimony must satisfy *Daubert* standards of reliability to be admissible.n33.10

[3] Reliance on Science and Medicine

[a] The Need for Expert Testimony. Historically, causation in fact did not present many difficult issues in tort litigation. There were, of course, issues involving preexisting conditions, intervening causes and mutual contributing causes. However, in the typical case the question of causation was left to the jury after submission of a minimal amount of evidence on the subject.n34 There rarely was a question that the traumatic injury observed immediately after the tortious conduct was caused by the conduct. Even if there was a question, it was normally a matter that could be resolved by the jury based on observational testimony.

In toxic tort litigation, however, causation is not a simple matter for the jury. The plaintiff must establish by a preponderance of evidence the presence of the injury-causing substance, that he or she has been exposed to the substance, and that the exposure has resulted in certain injuries.n35

The Reference Manual on Scientific Evidence sets forth the standard protocol used to extrapolate from a theoretical toxic risk to a real world conclusion about causation. First, the toxicologist should analyze whether the disease can be related to chemical exposure by a biologically plausible theory. Second, the expert should examine if the plaintiff was exposed to the chemical in a manner that can lead to absorption into the body. Finally, the expert should offer an opinion as to whether the dose to which the plaintiff was exposed is sufficient to cause the disease.n35.1 It is an axiom of toxicology that an assay of an individual's actual or probable exposure to a toxic agent "is essential in determining the effects of harmful substances."n35.2 Exposure can be measured or estimated on one of three ways: mathematical modeling, using direct measurements of the medium in question, or biological monitoring.n35.3

As will be discussed in greater detail throughout this chapter, establishing each of the required elements of causation presents significant problems to toxic tort plaintiffs.n36

It is a general rule that a jury is incapable of determining cause and effect relationships on scientific and medical matters without expert testimony.n37 This is because the causation inquiry in toxic tort cases is more complicated, as the injuries themselves are usually not immediately obvious, and the connection between exposure and injury is not a matter of common sense or everyday experience.n37.1 Thus, unlike traditional tort litigation, in which causal connections can be established by circumstantial evidence, toxic tort litigation, which relies so heavily on scientific evidence, requires that the causal chain be established by expert testimony. Therefore, it is not surprising that in toxic tort litigation a great proportion of the evidence will be testimony from expert witnesses.n38

When the defendant in a toxic tort case seeks summary judgment on the ground that there is no proof of causation, the plaintiff bears the burden of introducing evidence showing a reasonable basis for the conclusion that "it is more likely than not" that the conduct or product of the defendant was a cause-in-fact of the plaintiff's injuries.n38.1 A mere possibility of causation is insufficient,n38.2 and if the matter remains one of pure speculation or conjecture, or if the probabilities are at best evenly balanced, the court will grant the defendant's motion. In most jurisdictions, expert medical testimony is some evidence of causation if there is testimony of the reasonable probability of a causal connection between the act and the injury.n39 The expert need not use the exact magic words "reasonably medical probability;" the testimony is sufficient if circumstances show that this is the substance of the testimony. Expert testimony that is based on assumed facts that vary materially from actual undisputed facts is not evidence.n40 In South Carolina, expert testimony on the issue of causation must state that the result "most probably" came from the caused alleged.n40.1

In toxic tort cases, if no expert medical testimony is presented summary judgment will be granted.n41 For example, in a personal injury action brought as a result of exposure to toxins from a hazardous waste storage and disposal facility, summary judgment was entered for the defendants because the plaintiffs presented no expert medical testimony on the issue of causation, and the issue was outside the competence of the jury to determine.n42 Defendants prevailed for want of expert testimony in a case alleging injury from exposure to toxic compounds that emanated from window shades, as the potential harmful effects of chemical outgassing from a product manufactured with phenol is not within common knowledge.n43 Similarly, in a Nevada case in which the plaintiffs failed to present expert testimony on the issue of causation, the District Court for the District of Nevada granted summary judgment for the defendant.n44

The lack of consensus among expert witnesses will generally go to the weight of the testimony not its admissibility. Therefore, the fact that experts disagree will not result in summary judgment. For example, the Fifth Circuit upheld a $1.5 million verdict for the family of a deceased employee of the U.S. Forest Service who died of Hodgkins disease seven years after applying the herbicide 2,4-O in a weed control program. The Fifth Circuit upheld the admissibility of plaintiff's expert testimony on causation. The court ruled that the absence of scientific consensus went to the weight of the testimony, not its admissibility, as long as the expert relied on methods on which other experts would rely in forming opinions.n45

The Supreme Court of New Jersey held that a plaintiff in an occupational exposure, toxic tort case may demonstrate medical causation by establishing (1) factual proof of the plaintiff's frequent, regular and proximate exposure to a defendant's products; and (2) medical and/or scientific proof of a nexus between the exposure and the plaintiff's condition. The court stressed that the "frequency, regularity and proximity test" bears no relationship to theories of collective liability adopted by some courts, and assigns liability only to those defendants to whose products the plaintiff can demonstrate that he or she was intensely exposed.n46

[b] Law vs. Science; Probability vs. Certainty. It has long been recognized that law and science approach causation in fundamentally different ways.n47 Legal causation is a policy question as to when responsibility attaches because of the connection between the conduct and the result. The "but for" and the "substantial factor" rules, although called cause-in-fact tests, are policy resolutions as to the attachment of liability. In a very real sense, there are hundreds of causes of a given result but the law recognizes only some of the causes, and focuses on one or a limited number of acts as if the other events did not exist.n48 In addition, the law views the absence of events as causes. For example, the law recognizes that the failure to extinguish a fire may be as much a cause of the ultimate damage to a building as lighting the blaze in the first place.n49

Legal causation also is based on a preponderance of the evidence standard.n50 The plaintiff must establish that causation is more probable than not. Courts have equated this requirement with the determination of a probability exceeding 50 percent.n51 Moreover, the law approaches the problem of proof in an all-or-nothing fashion. If the plaintiff establishes that the probability of causation is at least 51 percent, then the plaintiff is entitled to recover for the full amount of his damages. On the other hand, if the plaintiff can only establish that the probability of causation is 49 percent instead of 51 percent, then the plaintiff does not recover at all.n52

Similarly, if the plaintiff does not present any evidence as to the percentage of cause attributable to the defendant, the plaintiff will not recover. For example, in *Hahn v. Weber & Sons Co.*,n53 a case for crop damage allegedly resulting from the drift of herbicide from a neighboring field, where drought presented an independent cause of the injury, the court ruled that the plaintiff had failed to meet his burden of establishing what percentage of the damage was attributable to the defendant.

Although some toxic tort plaintiffs have been able to establish that causation is more probable than not,this approach to proof has also resulted in the dismissal of many toxic tort cases. For example, in *Manko v. United States*,n54 the court found sufficient proof of causation of plaintiff's illness based on statistical evidence that the risk of contracting Guillain-Barre Syndrome 11 to 16 weeks after receiving a Swine Flu vaccine is 3.4 times greater than the risk among the non-vaccinated population. However, the District Court for the District of New Jersey, dismissed an action brought by an asbestos plaintiff who was only able to show through epidemiological data that he belonged to a class of persons facing a 43 percent chance of contracting cancer as a result of exposure to asbestos.n55 Subsequently, the New Jersey Supreme Court also upheld the dismissal of claims for increased risk of cancer from exposure to asbestos in the absence of a reasonable degree of medical certainty that the plaintiff was more likely than not to get cancer.n56

In a Maryland suit by workers claiming injury from a "toxic soup" in the workplace, the plaintiffs were unsuccessful because they could not prove a greater than 50 percent chance that any particular product caused their injuries. The District Court for the District of Maryland characterized the plaintiff's claims as a variant of a market share theory, which is not recognized in Maryland.n57

Some commentators have suggested alternatives to the "all or nothing" approach to causation, such as a valuation system that focuses on the loss of chance.n58 Plaintiffs have attempted to assert theories of proportional liability in toxic tort cases. In New Jersey, the Superior Court, Appellate Division, refused to apply the "lost chance" doctrine, in a case in which it was claimed that the plaintiff's mother died of pancreatic cancer as a result of exposure to dioxin from a chemical plant near her home. The "lost chance" doctrine reduces the plaintiff's burden of proving causation in certain cases in which the defendant's breach of a duty to the plaintiff. In order to prove causation, the plaintiff relied upon the testimony of a pathologist who stated that the decedent's risk of pancreatic cancer was substantially increased as a result of her exposure to dioxin. In rejecting the plaintiff's theory of causation, the court noted that acceptance of evidence of unquantified risk as proof of causation would generate a large number of cases that would be difficult for the courts to manage. Finally, the court held that in the absence of proof to a degree of reasonable medical probability that the defendant's release of dioxin caused the decedent's cancer, there was no liability.n59

Science, on the other hand, approaches causation not in terms of social responsibility but in terms of etiology. Scientists generate hypotheses or theories that are confirmed by rigorous standards of reproducibility and exclusion of other explanations.n60 Scientists may speak of associations and relationships, but it is difficult for them to use the term "cause" unless the probability approaches certainty. They test the strength of the association by statistical methods and are willing to call an association "causal" only when certain criteria are met, including consistency of association, correct time sequence, specificity of association, strength of association, and biological probability.

To scientists, statistical significance refers to the likelihood that an association is the result of pure chance.n61 The fact that the mathematical tests of statistical significance may be more stringent than the legal preponderance of evidence standard was discussed by Judge Jenkins in *Allen v. United States*.n62 Judge Allen also commented on the distinctions between causation in toxic tort cases and causation in traditional personal injury actions.n63 *Allen* was an action brought under the Federal Tort Claims Act to recover for incidences of cancer and leukemia allegedly caused by the Atomic Energy Commission's testing of atomic devices prior to 1933. In his opinion, Judge Jenkins concluded that whether a correlation between a cause and an effect was more likely than not a different question from that answered by tests of statistical significance, which often distinguish narrow differences in degree of probabilities.n64

The reluctance of scientists to specifically state that a causal relationship exists has, in some toxic tort cases, diminished the effectiveness of the expert's testimony. In a pesticide exposure case, the court struck portions of the plaintiffs' expert's affidavit which stated that the plaintiffs' health complaints were "not inconsistent with" exposure to the pesticide and could have been caused thereby as unsupported by a reasonable degree of scientific or medical certainty and insufficient to prove causation.n65

In other cases in which scientists were unable provide definitive testimony on the issue of causation, the plaintiffs' cases were dismissed. For example, in a case involving injuries from an IUD, the Fourth Circuit upheld a judgment for the defendant granted after trial on the grounds the plaintiffs was inadequate scientific proof of causation.n66 Similarly, the Fifth Circuit, applying Mississippi law, held that the district court properly excluded the affidavit of the plaintiff's expert because it lacked foundation and was unreliable. The expert stated that a link between asbestos exposure and the decedent's injury was statistically probable and could not be ruled out. In the absence of evidence of causation, the court found that the plaintiff failed to prove an essential element, and summary judgment was properly entered for the defendant.n67

In a Bendectin case, the Fifth Circuit, applying federal law, also reversed a district court's ruling denying the defendant's motion for a judgment notwithstanding the verdict. The circuit court was doubtful as to the reliability of the plaintiff's epidemiological evidence. Therefore, it ruled that the plaintiff's expert's testimony on causation was inadequate. The court concluded that the plaintiff's study did not demonstrate a statistically significant correlation between the drug and birth defects, that it was unreliable and that it had not been subject to peer review. The animal studies upon which the plaintiff relied were also flawed. The court encouraged district courts faced with medical and epidemiological evidence

in toxic tort cases to scrutinize the "basis, reasoning, and conclusiveness" of the studies, but stated that it did not intend to bar future Bendectin cases if new and conclusive studies demonstrated causation.n68

Scientists are uncomfortable with the legal standard of preponderance of evidence, since they often find it difficult to isolate one single factor as the cause of an event. To a scientist, imputing simple causation to one factor entails an artificial isolation or singling out of factors as though only X explains Y.n69

The inability of scientists to rule out "other causes" has been fatal to the claims of toxic tort plaintiffs.

It is a fundamental duty of a toxic tort lawyer to understand the difference between legal causation and scientific causation and to explain the difference to the potential expert witness. For a plaintiff's attorney, the problem is exacerbated by the use of the term "reasonable medical certainty." In most jurisdictions, it is understood that the term "reasonable medical certainty" does not mean scientific certainty but rather refers to the legal standard of preponderance of the evidence.n70

The courts have recognized that to require matters of medicine and science be demonstrable to a certainty would impose a greater burden of proof than is necessary. In a Louisiana action which arose when the plaintiffs developed respiratory and psychological problems after being exposed to chlorine gas, the trial court committed prejudicial error in concluding that the plaintiffs failed to prove cause-in-fact because they did not demonstrate by a "reasonable medical certainty" that the chlorine exposure caused the injuries. The court stated that in Louisiana, the plaintiff has the burden of proving every essential element of his case-including cause in fact-by a preponderance of the evidence. When the term "reasonable medical certainty" is used to describe the measure of persuasion in a tort case, it places upon the plaintiff a higher degree of proof than is required.n71

The basis for the admission and evaluation of scientific expert testimony has been the subject of confusion and disagreement among both courts and commentators.n72 This is a particularly "hot" issue in toxic tort cases because scientific evidence is such an integral part of any case involving exposure to toxic substances.

The courts have been struggling with the task of determining the standards for admissibility of scientific evidence. The original test for admissibility of scientific evidence was set forth in *Frye v. U.S.* n73 Under *Frye,* the standard for admissibility was "general acceptance in the scientific community."

Finally, in 1993, the issue of the admissibility of scientific evidence was addressed by the Supreme Court of the United States in the landmark case of *Daubert v. Merrell Dow Pharmaceuticals, Inc.*,n74 in which the court overruled the "general acceptance" test of *Frye*.n75 (*See* ch. 6, *Expert Witnesses*, for detailed discussions of the *Frye* and *Daubert* standards.)

[4] The Science of Causation

[a] Koch's Postulates. After Robert Koch developed and presented his postulates concerning the germ theory of disease in 1890, scientific developments continued. There were, even in Koch's lifetime, a number of infectious agents which did not meet all of his criteria, but which he felt were strongly implicated in the causation of disease. However, it was left to others to expand upon his original postulates. For example, attempts have been made to modify Koch's postulates to address possibilities not generally foreseen when the postulates were first enunciated, for instance, viruses as a cause of cancer or chronic central nervous system infection.n76

Koch expressed the requirements of his germ theory of causation in only three steps:

(1) The parasite occurs in every case of the disease and under circumstances which can account for the pathological changes and clinical course of the disease.

(2) It occurs in no other disease as a fortuitous and nonpathogenic parasite.

(3) After being fully isolated from the body and repeatedly grown in pure culture, it can induce the disease anew.

Koch's three requirements are based on logic. Unless the disease-bearing organism occurs in every case, and under circumstances related to pathological changes and the clinical course of the disease, the organism cannot be reliably linked to the disease process. If the parasite occurred in any other disease as a fortuitous and nonpathogenic parasite, then the scientist would be faced with the task of ruling out everything which was potentially causal of the other disease. Finally, if, after being fully isolated from the body and repeatedly grown in pure culture, the disease could not be reproduced, there would be no way to verify the correctness of the theory presented.

Koch's postulates, while admittedly not complete, lend themselves directly to modern experience dealing with chemicals. Flowing logically from the postulates is the underlying assumption that if their requirements are not satisfied, more evidence is required to insure the reliability of any hypothesis being examined. Obviously, opinions can be formulated on less evidence. The crucial issue is whether such opinions are both reliable and verifiable by other scientists. If not, such opinions properly remain classified only as hypotheses.

Even Koch's original postulates can be applied to diseases allegedly caused by chemicals. If a chemical exposure is not present in every case of the disease being studied, and under circumstances which account for all pathological changes during the course of the disease, no causal conclusions can be drawn. This is true because, with chemicals, an effect may be observed in an extremely small number of exposures. Further, with chemicals, there are often numerous confounding causes present. Finally, exposure to a chemical may not result in all the disease elements (pathological changes) being satisfied.

If the disease occurs under circumstances which might be defined as "idiopathic" in modern terms, then the second requirement of Koch's postulates is not met. A disease process is always potentially fortuitous (nonchemically related) as long as the possibility of an unknown cause remains open. Defense counsel should always present the contention that mere exposure to a chemical does not eliminate the idiopathic (unknown cause) possibility. A documented exposure to a chemical does not change the etiology of the disease automatically from "unknown" to "known." There must be a showing that there was an effect from the chemical on the person exposed, not merely that the chemical had the potential to do so or that the effect occurred after the exposure.n77

Logically, if there is a disease classification referred to as "idiopathic," then this implies that there is no known technique for attributing the disease process to any cause under certain circumstances. If 50 percent of the cases of aplastic anemia are idiopathic,n78 what circumstances permit the remaining 50 percent of the disease to be attributed to a cause? Good scientists will always concede that the largest portion of most disease "causes" remain in the unknown category.

There is no direct translation for the third requirement of Koch's postulates, because a chemical cannot be isolated from the body, grown in culture, and induce the disease anew. After being isolated from the body and repeatedly grown in culture, if the germ studied cannot induce the disease anew, then it has not been demonstrated to have the capability to induce the disease. The third requirement of Koch's postulates, as originally phrased, satisfied both the requirement that the disease organism be *capable* of causing the disease, as well as the cause-in-fact of a disease in a specific individual.

However, there is a logical parallel to Koch's third postulate where chemicals are involved. This is the requirement of epidemiological data. This is only a crude parallel, because epidemiological data only satisfies the "capability" requirement, not that of cause-in-fact. If the disease does not occur in the presence of a chemical with a greater frequency than in the absence of the chemical, then there is no evidence that the chemical has produced *any* effect in humans, at least where latent disease is concerned. Even when the capability of a chemical to cause a disease is established, the particular proof to establish cause-in-fact may not yet have been developed.

[b] Limits of Modern Science. When the symptoms of a latent disease occur only long after a chemical exposure, and when only a very small percentage of the population contracts the disease, *there is often no known scientific technique* for determining the cause of the disease in a specific individual. Modern science has not reached the point where it can reliably identify such a cause and effect relationship. What many scientists would term "speculation," however, plaintiffs' experts may elevate to "reasonable scientific certainty" in the courtroom. Defense counsel should not be hesitant to demonstrate to the court that modern science has not reached a point where such conclusions can be reliably drawn, and to vigorously challenge the methodology of plaintiff's experts when they attempt to argue the reliability of

their conclusions (see ch. 6, § 6.09[3], *above* , for a discussion with the reliability of expert testimony as a requirement to its admission into evidence).

Defense counsel can often effectively support the theme that modern science is inadequate to form reliable opinions as to the cause of most suspected chemically induced disease with concessions from the plaintiff's expert witness. Most experts will admit that after a disease has manifested itself, there is no clinical test available to determine the cause of a specific disease in a specific individual. They will further admit that while disease causation is a matter of inference to be deduced from the circumstances present in the case, there is an accepted scientific methodology for making such inferences.

[c] Defense Strategy: Focus on Requirement of Specificity of Causation Proof. The requirement of "specificity" of causation proof cannot be overemphasized. Both logically and scientifically, if one person in 100,000 develops aplastic anemia from causes completely unknown, before a chemical cause can be implicated there must be evidence presented demonstrating that the plaintiff is not that one in 100,000, and that the presence of a chemical exposure makes it more likely that the disease would have occurred than if there had been no such exposure. Dr. Alfred S. Evans discussed the requirement of the evidence needed to overcome this problem:n79

> The recognition of an association between smoking and lung cancer in 1950 and later of diet and cholesterol in relation to coronary heart disease stimulated efforts to draw up criteria for procedural and inferential methods in establishing causation. In a 1958 conference on this subject Drs. Yerushalmy and Palmer developed a set of suggested criteria. First they reviewed Koch's postulates and stressed that in view of their possible application to chronic disease two essential types of evidence related to (i) the simultaneous presence of the organism and disease and their appearance in the correct sequence and (ii) the specificity of the effect of the organism on the development of the disease. In particular, the need was stressed to establish the *specificity* of an effect in studies of chronic diseases. The concept of multiple causation was also emphasized in chronic disease as opposed to acute infectious disease.n80

Evans' two key requirements to implicate an agent as an etiologic factor in chronic disease where chemicals are suspected really boil down to the requirements for good epidemiology studies:

> (1) the suspected characteristic must be found more frequently in persons with the disease in question than in persons without the disease; or
>
> (2) persons possessing the characteristic must develop the disease more frequently than do persons not possessing the characteristic.

Evans then discusses additional factors for establishing causation where chronic disease is involved:

> (1) Incidence of the disease should increase in relation to the duration and intensity (dose) of the suspected factor.
>
> (2) The distribution of the suspected factor should parallel that of the disease in all relevant aspects.
>
> (3) A spectrum of illness should be related to exposure to the suspected factor.
>
> (4) Reduction or removal of the factor should reduce or stop the disease.
>
> (5) Human populations exposed to the factor in control studies should develop the disease more commonly than those not so exposed.n81

With some diseases, such as aplastic anemia or rarely occurring cancers, reduction of exposure to the factor in the general population can often be demonstrated not to have an effect on the overall incidence of the disease. Bendectin was one example where removal of the agent from the market had no effect on birth defects occurring in the nonexposed population.n82

Hackney and Linn provide another useful basis of analysis for the defense practitioner.n83 They assert:

> Although modern problems of ill health caused by toxic chemicals in the environment may appear to be less clear-cut than those faced by Koch's generation, they are not fundamentally different. The problem is still to demonstrate a cause-and-effect relationship between a particular entity in the environment and a particular health effect, with the goal of eliminating the hazard posed by the offending material after it has been identified.n84

It should be noted that while the authors correctly state that the problems posed by germ theory and chemical causation are not fundamentally different, they do not assert that the criteria can be fulfilled in the case of chemicals. Trial lawyers know from direct experience that in most cases such criteria are usually absent, and Hackney and Linn acknowledge this fact:

> Today, researchers must usually deal with specific health effects (e.g., dysfunction, disability, or aggravation of existing disease) and less well-defined environmental agents (e.g., mixtures of chemicals), thus their first postulate must accept less rigorous evidence (e.g., epidemiologically determined statistical associations between environmental and health variables or anecdotal clinical information).n85

What Hackney and Linn are saying to their scientific audience is that with chemicals, "particularistic" proof is often not achievable. The scientific world is forced to acknowledge less rigorous evidence which only establishes statistical association or the capability of a chemical to produce a particular effect. More specifically, scientists initially ask the question, "Is there an effect?" Often, the best answer available is only that there is a statistical association. Attorneys translate this into the statement, "Is the chemical capable of causing an effect?" Left unanswered is whether any given case involves a cause and effect relationship between the chemical and the disease.

Scientists recognize that an effect from the chemical may be direct or indirect. This distinction can be potentially valuable on deposition with an opposing expert witness, particularly where the witness asserts that the effect involved is "synergistic" or "additive." Counsel should keep in mind the fact that proof of a "synergistic" or "additive" effect requires the same type of proof that any other alleged chemical effect requires. If an effect is asserted for two chemicals acting together, then studies involving proof that those two chemicals in fact act together to produce an effect must be presented, as well as specific proof that they created a toxic response in the individual case.

While Hackney and Linn discuss Koch's Postulates and by inference rewrite those postulates to permit an application to laboratory testing, they do not present a definitive criterion which trial counsel can present to the court as a "unified concept" for sufficiency of evidence to establish an effect from a chemical. The best defense technique may be for counsel to paraphrase the Koch principles, and then obtain concessions of the validity of those principles from the plaintiff's experts on deposition. Many of the logical requirements of causal methodology are self-evident, and these concessions can be readily obtained. Defense experts, after a review of causation methodology, will invariably agree with defense counsel's presentation of "minimal" requirements to establish causation.

The central idea always is that *some methodology* must be presented to the court for its reference and use. As noted by Gots:

> It takes very little in the way of ill-founded testimony by a physician to support a jury's belief, despite the lack of any scientific validity for that belief. A conclusion about a cause and effect relationship must be supported by scientific evidence that such a relationship has been demonstrated and, therefore,

can occur. Otherwise it is not based upon reasonable, accepted scientific methodology and it is speculative and misleading to the trier of fact.n86

Gots, in his analysis of causation methodology, presents one of the most important concepts for a defense lawyer to keep in mind. He defines causation methodology as logically consisting of two kinds of evidence:

The proper principles of the methodology of causation analysis can be summarized as follows:

Can the agent in question produce the disease at issue?

(1) Is there substantial and properly relevant animal data?

(2) Is there human evidence, particularly epidemiological support?

Did it cause it in this case?

(1) Have other causes been properly considered and ruled out?

(2) Has the exposure been confirmed?

(3) Was the exposure sufficient in duration and concentration?

(4) Was the clinical pattern appropriate?

(5) Is the morphological pattern appropriate?

(6) Is the temporal relationship appropriate?

(7) Is the latency appropriate?n87

Gots's two-step methodology, although useful, creates an inferential trap for the defense attorney. The factors he discusses under, "Did it cause it in this case?" are not, by themselves, sufficient to prove cause in a particular case. The mere fact that steps one through seven of the Gots methodology have been fulfilled does not constitute "particularistic" proof. Steps one through seven are really additional requirements to prove causation capability. As a practical matter, the issue will never arise because the plaintiff in most cases will not have evidence to satisfy any causation elements, much less the additional considerations set forth by Gots.

The key to a correct scientific analysis also depends on an understanding of the "mechanism" of causation, i.e., "What does the chemical do to the body which results in an effect?" The questions to be asked in this regard are as follows:

(1) What specific response can be observed in humans given a specific level of exposure to the chemical?

(2) Can that response be observed as having occurred in the specific individual under observation?

(3) Is that response so unique that a coincidental effect can be ruled out?

Courts may never require a plaintiff to prove step three, but some minimal understanding of the mechanism of injury, coupled with that mechanism being demonstrated in the plaintiff, is a required part of "particularistic" proof. The re-

quirements of particularistic proof in this regard are not esoteric, but are derived from common sense, and can be understood by a court or jury from its own experience.

For example, with asbestosis, the presence of asbestos fibers in the lung tissue, coupled with the presence of necrotic tissue and the correct constellation of symptoms, "proves" the cause of the disease.n88 With vinyl chloride monomer, the appearance of angiosarcoma of the liver after high-level exposure to that chemical may prove a causal relationship simply because of the rarity of angiosarcoma of the liver.n89 However, the number of instances in which the effect will be so rare that a disease with an extremely long latency can be linked to a cause is exceedingly small. It should be emphasized that with vinyl chloride monomer, no information is known about low-level effects. The appearance of an angiosarcoma of the liver in the face of an exceedingly low exposure to vinyl chloride monomer would logically prove nothing to a scientist, because of the absence of evidence linking a low dose with the suspected cause. Speculation by an opposing expert does not supply the required evidence.

[5] A Unified Concept of Chemical Causation

The set of requirements listed below were developed by Haskell Shelton, Esq. to prove that a chemical is capable of causing an effect. These are the minimal elements required to prove scientifically that a chemical causes an effect. These steps follow a logical progression which is simple for the court to understand and apply to the particular facts of a case. When properly presented, the principles also illustrate that without fulfillment of the evidentiary requirements, existence of a causal relationship cannot be established. It cannot be overemphasized that these steps do not supply particularistic proof. *Even if* the plaintiff satisfies all of the steps in a given case, only evidence of the *capability* of a chemical to cause an effect has been proven:

(1) Exposure to the suspected cause (putative agent) must be documented (see § 10.02[1], *below*)

(2) The exposure must occur in such a way that the chemical is eligible to be the cause of the observed effect. For example, the chemical cannot be deemed the cause of an effect unless the effect is initiated after the chemical exposure.

(3) The documented level of exposure must be capable of inducing a known toxic effect.

(4) The observed toxic effect must be satisfactorily linked to some observed effect in a target organ.

(5) The suspected toxic effect must have been reproduced in the general human population upon identical exposure.

(6) Variables such as diseases induced by other drugs or chemicals, bacterial/viral causes, or other known causes must be eliminated.

(7) A consistent pattern of identical effects under controlled circumstances must be demonstrated (literature precedence).

(8) Epidemiologic and bioassay tests must be supportive.

Using these steps, it becomes apparent that the typical plaintiff's expert fails to meet most if not all of the required minimal evidentiary requirements. This permits defense counsel to argue that plaintiff's proof involves nothing more than an expert bringing his or her credentials and a totally unsupported opinion into the courtroom. Still required as "particularistic proof" is evidence of a mechanism of injury, and a linking of that chemical mechanism to the individual case. Also required is an explanation for differences in individual susceptibility which might affect the outcome in a specific case.

Finally, defense counsel should remember that in the face of equally plausible causes, no reliable scientific conclusions as to cause and effect can be drawn. By definition there are no scientific techniques to distinguish between the two equally plausible causes. Given differences in individual susceptibility to chemical exposures, a decision by a jury in the

face of equally plausible causes would obviously amount only to speculation. Case law in every state supports a directed verdict for the defense in such a situation.

FOOTNOTES:

(n1)Footnote 1. Becker v. Baron Bros., 138 N.J. 145, 151, 649 A.2d 613 (Sup. Ct. 1994) .

(n2)Footnote 2. Landrigan v. Celotex Corp., 127 N.J. 404, 413, 605 A.2d 1079 (Sup. Ct. 1992) .

(n3)Footnote 3. **Cases illustrating difficulty of proving medical causation in toxic tort cases.** James v. Bessemer Processing Co., 155 N.J. 279, 714 A.2d 898, 1998 N.J. LEXIS 625 (1998) (worker was exposed to chemicals over course of 26 years employment).

See, e.g.:

Alabama: Ex parte Diversey Corp., 742 So. 2d 1250, 1254 (Ala. 1999) .

California: Bockrath v. Aldrich Chem. Co., 21 Cal. 4th 71, 86 Cal. Rptr. 2d 846, 980 P.2d 398 (1999) (plaintiff, who contracted multiple myeloma, named 55 defendants, alleging that disease arose through exposure to harmful substances in their products; in ordinary personal injury lawsuit, it suffices to plead causation succinctly and generally; in contrast, when pleaded facts do not naturally give rise to inference of causation, plaintiff must plead specific facts; although complaint was poorly drafted, court understood plaintiff to be attempting to allege that defendants' products cause cancer, and allegations were insufficient, court remanded case to give plaintiff opportunity to allege that each defendant's product was substantial factor in causing his multiple myeloma).

Williams v. NGF, Inc., 994 S.W.2d 255, 1999 Tex. App. LEXIS 3555, 99:20 Tex. Civil Op. Serv. 147 (plaintiff noticed pesticide-like aroma after defendant supplied store with flowers; defendant was entitled to summary judgment where testimony of her treating physician that volatile chemicals were most common cause of reactive airway disease (RAD), and that plaintiff was not tested for reactivity for specific substances because facilities were not available, provided no evidence of causal connection between plaintiff's RAD and chemicals used by defendant other than possibility of such connection).

(n4)Footnote 3.1. *See, e.g.,* Eggen, *Toxic Torts, Causation, and Scientific Evidence After Daubert,* 55 U. Pitt. L. Rev. 889 (Spring 1994); Voke, *Sources of Proof of Causation in Toxic Tort Cases,* 61 Def. Couns. J. 45 (Jan. 1994); Stundtner, *Proving Causation: T Cell Studies,* 20 Boston College of Envtl. Affairs L. Rev. 335 (Winter 1993); Callaghan, *Establishment of Causation in Toxic Tort Litigation,* 23 Ariz. St. L.J. 605 (1991); Jones, *Problems of Causation in Toxic Torts,* 55 Def. Couns. J. 282 (1988); Whitehead & Espel, *Legal Proof of Causation in Toxic Tort Litigation,* 2 Toxics Rep. 1040 (1988); N. Orloff, *Theories of Cancer and Rules of Causation,* 27 Jurimetrics 255 (Spring 1987); Kanner, *Emerging Conceptions of Latent Personal Injuries in Toxic Tort Litigation,* 18 Rutgers L. J. 343 (1987); Roisman, *Proving Cause in Toxic-Tort Litigation: The Threshold of a New Era,* 22 Trial 59 (1986); Keeton, *Causation,* 28 Tex. L. Rev. (1986); Harris, *Toxic Tort Litigation and the Causation Element: Is There Any Hope of Reconciliation,* 40 Sw. L. J. 909 (1986); Rosenberg, *The Causal Connection In Mass Exposure Cases: A "Public Law" Vision of the Tort System,* 97 Harv. L. Rev. 851 (1984); Note, *Establishing Causation In Chemical Exposure Cases: The Precursor Symptoms Theory,* 35 Rutgers L. Rev. 163 (1983); Trauberman, *Statutory Reform of "Toxic Torts": Relieving Legal, Scientific, and Economic Burdens on the Chemical Victim,* Environmental Law Institute (Sept. 1982); Trauberman, *Compensating Victims of Toxic Substance Pollution: An Analysis of Existing Federal Statutes,* 5 Harv. Envtl. L. Rev. 1 (1981).

(n5)Footnote 4. Superfund Section 301(e) Study Group, 97th Cong., 2d Sess., Injuries and Damages From Hazardous Wastes-Analysis and Improvements of Legal Remedies (Comm. Print 1982).

(n6)Footnote 4.1. Dreyer, *An Epidemiologic View of Causation: How It Differs From the Legal,* 61 Def. Couns. J. 40 (Jan. 1994); James, *Role of Toxicology in Toxic Tort Litigation: Establishing Causation,* 61 Def. Couns. J. 28 (Jan. 1994); National Research Council, Environmental Epidemiology, Public Health and Hazardous Wastes (1991); National Science Foundation, Compensation for Victims of Toxic Pollution-Assessing the Scientific Knowledge Base (1983); Davis, Bridbord & Schneiderman, *Estimating Cancer Causes: Problems in Methodology, Production, and Trends,* in Banbury Report 9: Quantification of Occupational Cancer 228 (R. Peto & M. Schneidmor eds. 1981).

(n7)Footnote 4.2. **Trend towards relaxed standard of medical causation in toxic tort cases.** Vassallo v. American Coding & Marking Ink Co., 345 N.J. Super. 207, 784 A.2d 734, 739 (2001) (expert reports submitted by

plaintiff who claimed that exposure to Resisto marking ink caused physical and psychiatric disorders established prima facie case of medical causation).

(n8)Footnote 4.3. *See, e.g.,* Smith v. Johnson, 313 P.2d 7, 8 (1957) .

See 5 Personal Injury: Actions, Defenses, Damages, *Negligence* (Matthew Bender).

(n9)Footnote 5. *See* 1 Damages in Tort Actions (Matthew Bender).

See 5 Personal Injury: Actions, Defenses, Damages, *Negligence* (Matthew Bender).

(n10)Footnote 6. For a discussion of causation in fact *see* Eggen, *Toxic Torts, Causation, and Scientific Evidence After Daubert,* 55 U. Pitt. L. Rev. 889, 895-897 (Spring 1994).

(n11)Footnote 7. *See* Schoolmaker v. Kaltenbach, 236 Wis. 138, 294 N.W. 794, 1940 Wisc. LEXIS 337 .

See W. Prosser and W. Keeton, *above* note 4, at 266.

See generally A. Becht & F. Miller, The Test of Factual Causation, 13-21 (1961).

(n12)Footnote 8. W. Prosser and W. Keeton, *above* note 4, at 266.

(n13)Footnote 9. *See* Anderson v. Minneapolis, St. Paul & Sault Ste. Marie Railway Co., 146 Minn. 430, 179 N.W. 45, 49 (1920) .

California:

Hoeffer v. Rockwell Automation, Inc., A107353, 2006 Cal. App. Unpub. LEXIS 701, at *9-*22 (Jan. 26, 2006) (not to be published) (evidence supported jury's finding that exposure to defendant's asbestos-containing products was a substantial factor in contributing to aggregate dose of asbestos plaintiff inhaled or ingested, and hence to risk of developing asbestos-related cancer).

(n14)Footnote 9.1. **Cause must be sufficient under substantial factor test.** Aldridge v. Goodyear Tire & Rubber Co., 34 F. Supp. 2d 1010, 1020 (D. Md. 1999) , applying Maryland law (where defendant supplied only 10% of 200 hazardous chemicals to which plaintiffs were exposed during course of employment at tire manufacturing plant, and experts were not able to isolate toxic effects of defendant-supplied chemicals, plaintiffs failed to show that any particular, identifiable defendant-supplied chemical was of itself sufficient to cause harm).

(n15)Footnote 10. Anderson v. Minneapolis St. Paul & Ste. Marie Railway Co., 146 Minn. 430, 179 N.W. 45, 49 (1920) ; *see also* Mavroudis v. Pittsburgh-Corning Corp., 86 Wash. App. 22, 935 P.2d 684, 689 (1997) (multi-supplier asbestos injury cases call for substantial factor test of causation rather than but-for test).

(n16)Footnote 11. *See* Restatement (Second) of Torts, § 433B, comment a, subsec. 1. *See also* Sheffield v. Owens-Corning Fiberglass Corp., 595 So. 2d 443, 1992 Ala. LEXIS 113, 1993 A.M.C. 2338, CCH Prod. Liab. Rep. P13135 , citing Restatement (Second) of Torts, § 431.

(n17)Footnote 12. Diel v. Flintkote Co., 204 A.D.2d 53, 611 N.Y.S.2d 519, 521 (1994) .

(n18)Footnote 13. Sheffield v. Owens-Corning Fiberglass Corp., 595 So. 2d 443, 1992 Ala. LEXIS 113, 1993 A.M.C. 2338, CCH Prod. Liab. Rep. P13135 , applying federal maritime law. This was an action brought under the Jones Act by merchant seamen who suffered from asbestosis.

(n19)Footnote 14. MCIC, Inc. v. Zenobia, 86 Md. App. 456, 587 A.2d 531, 540 (1991) .

(n20)Footnote 15. *See, e.g.,* Blair v. Eagle-Picher Indus., Inc., 962 F.2d 1492, 1497 (10th Cir. 1992) .

(n21)Footnote 16. *See, e.g.,* In re Compensation of Benson, 101 Or. App. 122, 789 P.2d 694, 696 (1990) .

(n22)Footnote 17. Blair v. Eagle-Picher Indus., Inc., 962 F.2d 1492, 1497-1498 (10th Cir. 1992) .

(n23)Footnote 18. Cimino v. Raymark Indus. Inc., 751 F. Supp. 649, 654 (E.D. Tex. 1990) , applying Texas law.

(n24)Footnote 19. Theer v. Phillip Carey Co., 133 N.J. 610, 628 A.2d 724, 728-729 (1992) .

(n25)Footnote 20. Thacker v. UNR Indus., 151 Ill. 2d 343, 177 Ill. Dec. 379, 603 N.E.2d 449, 455 (1992).

(n26)Footnote 21. Benshoof v. National Gypsum Co., 761 F. Supp. 677, 679 (D. Ariz. 1991), applying federal and Arizona law.

(n27)Footnote 22. Robertson v. Allied Signal, Inc., 914 F.2d 360, 366-367 (3d Cir. 1990), applying Pennsylvania law. This was an action to to determine the role of expert testimony and the "fiber drift" theory in proving causation in asbestos cases

(n28)Footnote 23. Robertson v. Allied Signal, Inc., 914 F.2d 360, 366-367 (3d Cir. 1990).

(n29)Footnote 24. Tragarz v. Keene Corp., 980 F.2d 411, 420 (7th Cir. 1992), an asbestos action applying Illinois law.

(n30)Footnote 25. *See also* Shine v. Owens-Illinois, Inc., 979 F.2d 93, 97 (7th Cir. 1992), applying Illinois law.

(n31)Footnote 26. Owens-Illinois v. Armstrong, 87 Md. App. 699, 591 A.2d 544, 1991 Md. App. LEXIS 140, CCH Prod. Liab. Rep. P12937.

(n32)Footnote 27. *See also* Owens-Illinois v. Armstrong, 326 Md. 107, 604 A.2d 47, 52-53 (1992).

(n33)Footnote 28. Maryland, Gist v. Raymark Indus., No. 86-309045 (Md. Cir. Ct. March 15, 1988).

(n34)Footnote 29. Welch v. Keene Corp., 31 Mass. App. Ct. 157, 575 N.E.2d 766, 769-770 (1991).

(n35)Footnote 30. Money v. Manville Corp. Asbestos Disease Comp. Trust Fund, 596 A.2d 1372, 1375 (Del. 1991).

(n36)Footnote 31. *See also* Threadgill v. Armstrong World Indus., Inc., 928 F.2d 1366, 1376-1377 (3d Cir. 1991), applying federal and Delaware law.

(n37)Footnote 32. *See, e.g.,* Thurston v. U.S., 888 F. Supp. 1100, 1109 (D. Utah 1995).

(n38)Footnote 33. *See, e.g.,* Reynard v. NEC Corp., 887 F. Supp. 1500, 1505 (M. D. Fla. 1995).

(n39)Footnote 33.1. **General causation defined.** *See* In re Hanford Nuclear Reservation Litig. (Jaros v. E.I. Du-Pont), 292 F.3d 1124, 1134 (9th Cir. 2002).

(n40)Footnote 33.2. **General and specific causation.** In re Breast Implant Litig., 11 F. Supp. 2d 1217, 1224 (D. Colo. 1998).

9th Circuit:

Whisnant v. United States, No. C03-5121FDB, 2006 U.S. Dist. LEXIS 80312, at *6 (W.D. Wash. Oct. 24, 2006) (plaintiff who claimed injuries caused by exposure to mold failed to create an issue of genuine material fact as to either general or specific causation).

Ohio:

Terry v. Ottawa County Bd. of Mental Retardation & Dev. Delay, 2006 Ohio 866, 2006 Ohio App. LEXIS 776, at *49 (air sampling reports and plaintiffs' depositions was sufficient to raise genuine issue of material fact regarding specific causation in toxic mold case).

But see Kreutzer v. Metabolife Int'l, Inc., D040651, 2004 Cal. App. Unpub. LEXIS 2242, at *11 n.4 (Mar. 11, 2004) (unpublished) (noting that no published decision by California state court has applied general/specific dichotomy in discussing element of causation).

(n41)Footnote 33.3. **Careful scrutiny is required when expert uses different substance to prove general causation.** *See, e.g.:*

11th Circuit:

Rider v. Sandoz Pharms. Corp., 295 F.3d 1194, 1201 (11th Cir. 2002) (Parlodel).

Benkwith v. Matrixx Initiatives, Inc., 467 F. Supp. 2d 1316, 1327 (M.D. Ala. 2006) (expert attempted to show that zinc gluconate has same effect on olfactory epithelium as zinc sulfate).

(n42)Footnote 33.3. **Proof of general causation may not be required in sporadic accident model cases.** Kuhn v. Sandoz Pharms. Corp., 14 P.3d 1170, 1184-1185 (Kan. 2000) (circumstances distinguished case involving drug Parlodel from cases that have employed general causation requirement).

(n43)Footnote 33.3.1. **Proving specific causation.** Merrell Dow Pharms. v. Havner, 953 S.W.2d 706, 1997 Tex. LEXIS 67, 40 Tex. Sup. Ct. J. 846, CCH Prod. Liab. Rep. P15015 .

(n44)Footnote 33.3.2. **Plaintiff must prove actual level of exposure.** Wright v. Willamette Indus., 91 F.3d 1105, 1996 U.S. App. LEXIS 18922, 45 Fed. R. Evid. Serv. (CBC) 377 .

(n45)Footnote 33.4. 45 U.S.C.S. §§ 51-60.

(n46)Footnote 33.5. **FELA has relaxed standard of proof regarding causation.** Bowe v. CONRAIL, 2000 U.S. App. LEXIS 24866 (not recommended for full-text publication), applying FELA (because railroad employee allegedly suffered multiple physical injuries as result of chemical exposure, jury could find from medical report, in context with plaintiff's own testimony and doctor's report, that Conrail's use of toxic chemicals contributed to his illness).

(n47)Footnote 35.5.1. **Employer is not insurer of employees' safety.**

Schrum v. Burlington Northern & Santa Fe Ry Co., No. Civ 04-619 PHX RCB, 2006 U.S. Dist. LEXIS 31995, at *6-*10 (D. Ariz. May 17, 2006), applying FELA (plaintiff presented insufficient evidence that inhalation of lime, coal, and coke dust caused his respiratory ailments, depression, and OCD).

Hamilton v. CSX Transp., Inc., No. CV504-12, 2006 U.S. Dist. LEXIS 48082, at *11-*13 (S.D. Ga. July 14, 2006) , applying FELA (no evidence that decedents' alleged exposure to asbestos caused their deaths).

(n48)Footnote 33.6. **Expert testimony is generally required in toxic tort FELA cases.** Denton v. Northeast Ill. Reg. Commuter. R.R. Corp., No. 02C2220, 2005 U.S. Dist. LEXIS 12773 , at *11-*16 (N.D. Ill. June 16, 2005), applying FELA (granting defendant's motion for summary judgment in absence of admissible evidence of causal link between her illness and mold and fungus contamination in her workplace); Coho v. CONRAIL, 2005 U.S. Dist. LEXIS 37081 , applying FELA (granting defendant's motion for summary judgment where plaintiff did not identify an expert to testify that his alleged workplace exposures to various toxins caused his injuries; Fulmore v. CSX Transp., Inc., 252 Ga. App. 884, 557 S.E.2d 64, 72-75 (2001) , applying FELA (evidence was sufficient to establish that plaintiffs who contracted asbestosis had been exposed to dangerous level of asbestos, without regard to specific level of asbestos contained in work environment).

But see:

Ufik v. Metro-North Commuter R.R., 77 F.3d 54, 59-60 (2nd Cir. 1996) (expert testimony was not necessary in case by plaintiff injured in fall because trier of fact could reasonably determine on its own that prolonged exposure to paint fumes would cause dizziness).

(n49)Footnote 33.7. **Daubert standard applies in FELA cases.** Mo. Pac. R.R. Co. v. Navarro, 2002 Tex. App. LEXIS 4533), applying FELA (expert testimony offered to prove that exposure to diesel exhausst causes myltiple myeloma was unreliable).

(n50)Footnote 33.8. **Court clarified standard for causation.** Gautreaux v. Scurlock Marine, 107 F.3d 331; 1997 U.S. App. LEXIS 3910 .

See also:

Torrejon v. Mobil Oil Co., 2003-1426 (La. App. 4 Cir. 06/02/04), 876 So. 2d 877, 886-888 , applying Jones Act (because parties stipulated that seaman's mesothelioma was caused by asbestos exposure, jury's determination that Mobil did not cause his injuries was completely devoid of support, thereby warranting plaintiff's JNOV motion).

(n51)Footnote 33.9. 46 U.S.C.S. § 688.

(n52)Footnote 33.10. **Expert testimony on issue of causation may be necessary in Jones Act cases.**

Wills v. Amerada Hess Corp., 379 F.3d 32, 41 (2nd Cir. 2004) , applying Jones Act, general maritime law, and New York law (affirming finding that plaintiff failed to satisfy burden of establishing causal link between vessels' alleged toxic emissions and decedent's illness and rejecting contention that because suit was brought under Jones Act, burden-shifting rule prescribed by *The Pennsylvania,* 86 U.S. (19 Wall.) 125, 22 L. Ed. 148 (1873) should apply, and the standards of reliability of Daubert should be relaxed).

(n53)Footnote 34. Personal Injury: Actions, Defenses, Damages, *Negligence* (Matthew Bender 1982).

(n54)Footnote 35. *See, e.g.,* Eggen, *Toxic Torts, Causation, and Scientific Evidence After Daubert,* 55 U. Pitt. L. Rev. 889 (Spring 1994); Voke, *Sources of Proof of Causation in Toxic Tort Cases,* 61 Def. Couns. J. 45 (Jan. 1994); Stundtner, *Proving Causation: T Cell Studies,* 20 Boston College of Envtl. Affairs L. Rev. 335 (Winter 1993); Callaghan, *Establishment of Causation in Toxic Tort Litigation,* 23 Ariz. St. L. J. 605 (1991); Jones, *Problems of Causation in Toxic Torts,* 55 Def. Couns. J. 282 (1988); Whitehead & Espel, *Legal Proof of Causation in Toxic Tort Litigation,* 2 Toxics L. Rep. 1040 (1988) ; Rosenberg, *The Causal Connection In Mass Exposure Cases: A "Public Law" Vision of the Tort System,* 97 Harv. L. Rev. 851 (1984); Note, *Establishing Causation In Chemical Exposure Cases: The Precursor Symptoms Theory,* 35 Rutgers L. Rev. 163 (1983); Trauberman, *Statutory Reform of "Toxic Torts": Relieving Legal, Scientific, and Economic Burdens on the Chemical Victim,* Environmental Law Institute (Sept. 1982); Trauberman, *Compensating Victims of Toxic Substance Pollution: An Analysis of Existing Federal Statutes,* 5 Harv. Envtl. L. Rev. 1 (1981).

(n55)Footnote 35.1. Reference Manual on Scientific Evidence, at 201 (Fed. Jud. Center 1994), *available at* http://www.fjc.gov/EVIDENCE/science/sc_ev_sec.html.

(n56)Footnote 35.2. Reference Manual on Scientific Evidence, at 206 (Fed. Jud. Center 1994).

(n57)Footnote 35.3. Reference Manual on Scientific Evidence, at 206 (Fed. Jud. Center 1994).

See Polaino v. Bayer Corp., 122 F. Supp. 2d 63, 70-71 (D. Mass. 2000) (plaintiff's expert could not prove that plaintiff was exposed to toxic agent, must less than he was exposed to sufficient dose to produce RADS response).

(n58)Footnote 36. Mason v. Texaco, Inc., 741 F. Supp. 1472, 1484 (D. Kan. 1990) , applying federal and Kansas law.

Goewey v. United States, 886 F. Supp. 1268, 1279 (D.S.C. 1995) , applying South Carolina law. An action was brought on behalf of an infant who allegedly sustained neurotoxic effects four months after being exposed to roof sealant. When a medical causal relation issue is not within the common knowledge of laypersons, proximate cause cannot be determined without expert testimony. Here, the opinion of the infant's physician as to causation was not sufficiently definite to be admissible.

(n59)Footnote 37. **Jury needs expert testimony to determine cause and effect in scientific and medical matters.** *See:*

Willert v. Ortho Pharmaceutical Corp., 995 F. Supp. 979, 983 (D. Minn. 1998) , applying Minnesota law (court excluded expert's opinion that it was probable to reasonable degree of medical certainty that Guillain-Barre Syndrome (GBS) was causally related to her treatment with Floxin; where question involves obscure and abstruse medical factors such that ordinary layman cannot reasonably possess well-founded knowledge of the matter, there must be expert testimony that thing alleged to have caused result not only might have done so, but in fact did cause resul);

Downs v. Perstorp Components, Inc., No. 3:96-cv-597, 2000 U.S. Dist. LEXIS 20281, at *10-*11 (E.D. Tenn. March 15, 2000) , applying Tennessee law (without expert testimony, plaintiff could not prove that one-time exposure to chemical Rubiflex caused illnesses from which he claimed to suffer).

Stadish v. Southern Cal. Gas Co., B1457579, 2002 Cal. App. Unpub. LEXIS 5648, at *21-*23 (June 24, 2002) (not to be published) (in toxic tort case, expert testimony is required to prove, with reasonable medical probability, that exposure to the defendant's products or emissions-here, benzene-caused plaintiff's injuries).

Nichols v. Three Bond of Am., Inc., CV990079127, 2001 Conn. Super. LEXIS 18641-*2 (July 10, 2001) (unreported) (where plaintiff claimed allergic reaction to chemical product, issue of fact remained as to claim of failure to warn and need for expert testimony).

Dunham v. Village of Canisteo, 303 N.Y. 498, 104 N.E.2d 872 (1952) .

Coastal Tankships, U.S.A., Inc. v. Anderson, No. 01-99-01345-CV, 2002 Tex. App. LEXIS 4091, at *26-*27 (May 31, 2002), applying general maritime law (expert testimony was required to prove that exposure to naphtha caused bronchiolitis obliterans organizing pneumonia (BOOP), which has several possible causes and is often idiopathic).

(n60)Footnote 37.1. **Causation inquiry in toxic tort cases is complicated.** In re Meridia Prods. Liab. Litig., 328 F. Supp. 2d 791, 798 (N.D. Ohio 2004) , applying Ohio law.

(n61)Footnote 38. *See* ch. 6, *Expert Witnesses*; *see generally* S. Baldwin, Art of Advocacy: Direct Examination (Matthew Bender 1986); Smith, *Admissibility of Expert Testimony and the Toxic Tort*, 15 J. Prod. and Tox. Liab. 97 (Spring 1993).

(n62)Footnote 38.1. **Plaintiff's burden of proof in response to summary judgment motion.** Armstrong v. Durango Paper Co., No. CV202-085, 2005 U.S. Dist. LEXIS 22366, at *16 (S.D. Ga. Sept. 27, 2005), applying Georgia law.

(n63)Footnote 38.2. **Mere possibility of causation is insufficient.** Hudjohn v. S & G Mach. Co., 200 Ore. App. 340, 114 P.3d 1141, 1149 (2005) . Although "magic words" are not required, an expert's opinion, read as a whole, must establish a probability of causation. Here, the expert's testimony that inhalation of toxins may have accounted for some degree of plaintiff's neuropsychological deficits was explicitly stated in terms of possibility, not probability.

(n64)Footnote 39. **"Reasonable medical probability" standard.** *See, e.g.:*

California:

Meza v. H. Muehlstein & Co., 2007 Cal. App. Unpub. LEXIS 458, at *48-67 (Jan. 19, 2007) (not to be published) (experts provided sufficient foundation for their opinions that occupational exposure to plastic dust and fumes caused plaintiff's respiratory illnesses).

Texas:

Baxter Healthcare Corp. v Grimes, No. 05-95-01682-CV, 1998 Tex. App. LEXIS 5505, at *4 (Aug. 31, 1998) (unpublished) (silicone breast implants).

(n65)Footnote 40. Baxter Healthcare Corp. v Grimes, No. 05-95-01682-CV, 1998 Tex. App. LEXIS 5505, at *4 (Aug. 31, 1998) (unpublished) (testimony of rheumatologist, professor of pharmacology, and neurologist constituted sufficient evidence that silicone breast implants caused plaintiff's illness).

(n66)Footnote 40.1. **Standard for expert testimony in South Carolina.** Clark v. Greenville Co., 313 S.C. 205, 437 S.E.2d 117, 119 (1993) (expert admitted to merely assuming that landfill was the source of contamination).

See Goewey v. United States, 886 F. Supp. 1268, 1995 U.S. Dist. LEXIS 6992, 33 Fed. R. Serv. 3d (Callaghan) 1020 , applying South Carolina law.

(n67)Footnote 41. **Defendant is entitled to summary judgment in toxic tort cases in absence of expert testimony.** *See, e.g.:*

3rd Circuit:

Amico v. Duracal Cement, No. 04-4924(WGB), 2006 U.S. Dist. LEXIS 55329, at *12-*18 (D.N.J. Aug. 8, 2006) (not for publication) (granting summary judgment in favor of manufacturer in absence of admissible expert testimony that exposure to quick-drying cement caused achalasia).

5th Circuit:

Dupree v. Valero Energy Corp., 2005 U.S. App. LEXIS 17930, at *5-*6 (5th Cir. 2005) (unpublished) (defendant was properly granted summary judgment where plaintiff offered no expert testimony to support contention that work-place exposure to gasoline fumes with chemical additive t-amyl methyl ether exacerbated his gastro esophageal reflux disease, hiatal hernia, and hemorrhoids, resulting in bilateral maxillary sinus disease).

Smith v. ADT Sec. Servs., 2006 U.S. Dist. LEXIS 70109, at *12 (S.D. Miss. Sept. 26, 2006) (dismissing personal injury and emotional distress claims in absence of expert testimony that mold exposure caused plaintiff's erectile dys-function, bloody urine, and diabetes).

10th Circuit:

Koch v. Shell Oil Co., 49 F. Supp. 2d 1262, 1266-1267 (D. Kan. 1999) (defendant was entitled to summary judg-ment in action alleging that feed addictive caused death of dairy herd and physical injuries to plaintiff's family where there was insufficient evidence to link Rabon with plaintiffs' medical conditions).

Willert v. Ortho Pharmaceutical Corp., 995 F. Supp. 979, 981-983 (D. Minn. 1998) , applying Minnesota law (fact that GBS followed ingestion of Floxin did not prove causation; moreover, theory was not tested or subject to peer re-view; there was no peer literature, and witness did not show that theory was generally accepted by scientific commu-nity; because court excluded testimony of plaintiff's expert, he was unable to establish causation, and defendants were entitled to summary judgment).

Kolesar v. United Agri Prods., Inc., 412 F. Supp. 2d 686, 696-698 (W.D. Mich. 2006) applying Wisconsin law (lack of expert testimony entitled defendant to summary judgment in action contending that RADS was caused by exposure to the liquid fertilizer metam sodium).

Bryant v. Metric Property Mgmt., No. 4:03-CV-212-Y, 2004 U.S. Dist. LEXIS 11214, at *20-*21 (N.D. Tex. June 17, 2004) (applying Texas law) (mold).

9th Circuit:

Whisnant v. United States, No. C03-5121FDB, 2006 U.S. Dist. LEXIS 80312, at *6 (W.D. Wash. Oct. 24, 2006) (in absence of expert testimony, plaintiff who claimed injuries caused by exposure to mold at commissary meat department failed to create an issue of genuine material fact as to either general or specific causation).

Montana:

Nelson v. Nelson, 329 Mont. 85, 2005 MT 263, 122 P.3d 1196, 1202-1203 (2005) . Plaintiff alleged that she was exposed to various chemicals as a result of her former husband's improper pesticide application at the ranch he operated. Without any expert testimony regarding causation, she could not prevail on her negligence claim).

New York:

Coratti v. Wella Corp., 2006 N.Y. Misc. LEXIS 3790, *23, 2006 NY Slip Op 52409U (N.Y. Sup. Ct. Dec. 15, 2006) (unpublished) (dismissing complaint in absence of admissible testimony that exposure to chemicals in defendant's hair coloring products could cause MCS, COPD, and other ailments).

Ohio:

Braglin v. Lempco Indus., 2007 Ohio 1964, 2007 Ohio App. LEXIS 1773, at *14-16 (2007) (without expert testi-mony, plaintiff was unable to establish proximate cause in intentional tort action alleging that decedent's exposure to solvents caused pancreatic cancer).

See also:

Bernhardt v. Richardson-Merrell, Inc., 892 F. 2d 440, 443-444 (5th Cir. 1990) (Bendectin).

Conde v. Velsicol Corp., 804 F. Supp. 972, 1021-1022 (S.D. Ohio 1992) , applying Ohio law.

Money v. Manville Corp. Asbestos Disease Comp. Trust Fund, 596 A.2d 1372, 1377 (Del. 1991) (asbestos).

Nitecki v. National Fuel Gas Dist. Corp., 193 A.D.2d 1098, 598 N.Y.S.2d 411, 412 (1993) (plaintiff's allegation that she had been injured as result of carbon monoxide exposure, without supporting expert opinion, was insufficient to defeat defendant's motion for summary judgment).

But see:

Rietcheck v. City of Arlington, 04-CV-1239-BR, 2006 U.S. Dist. LEXIS 30265, at *10-*11 (D. Or. May 15, 2006), applying Oregon law. Plaintiffs provided evidence only that certain water pipes contained elevated levels of hexavalent chromium one year before they became ill, and no evidence that defendants contaminated their tap water with hexavalent chromium or that they actually ingested the chemical. However, the jury could reasonably infer without expert testimony that defendants introduced some kind of contaminant into plaintiffs' tap water and that they became ill after being exposed to that contaminated water, and defendants were not entitled to summary judgment on the more generic causation issue.

(n68)Footnote 42. Paris v. Chemical Waste Management Inc., No. CV 86-2312 W (N.D. Ala. Dec. 7, 1987).

(n69)Footnote 43. **Lack of expert testimony entitled manufacturer to summary judgment.** Alden v. Phifer Wire Prods., 2005 Ohio App. LEXIS 2864, at *7.

(n70)Footnote 44. Layton v. Yankee Caithness Joint Venture, L.P., 774 F. Supp. 576, 580 (D. Nev. 1991) , applying Nevada law.

(n71)Footnote 45. Peteet v. Dow Chem. Co., 868 F.2d 1428, 1433-1434 (5th Cir. 1989) , applying federal law.

(n72)Footnote 46. James v. Bessemer Processing Co., 155 N.J. 279, 714 A.2d 898, 1998 N.J. LEXIS 625 (July 27, 1998) (worker was exposed to chemicals over course of 26 years employment).

(n73)Footnote 47. *See* Dreyer, *An Epidemiologic View of Causation: How It Differs From the Legal*, 61 Def. Couns. J. 40 (Jan. 1994); James, *Role of Toxicology in Toxic Tort Litigation: Establishing Causation*, 61 Def. Couns. J. 28 (Jan. 1994); Dannen & Sagill, *Medicolegal Causation: A Source of Professional Misunderstanding*, 3 Am. J. L. & Med. No. 3, 302 (1977).

(n74)Footnote 48. W. Prosser and W. Keeton, note 4, *above*, at 266.

(n75)Footnote 49. *See* Lee v. Carwile, 168 So. 2d 469, 473 (1964) .

See also W. Prosser and W. Keeton, note 4, *above*, at 266-267; 5 Personal Injury: Actions, Defenses, Damages, *Negligence* (Matthew Bender 1982).

(n76)Footnote 50. *See* W. Prosser and W. Keeton, note 4, *above*, at 239.

See, e.g., Santosky v. Kramer, 455 U.S. 745, 755, 102 S. Ct. 1388, 71 L. Ed. 2d 599 (1982) , in which the Supreme Court held that the preponderance of the evidence standard was sufficient in most civil matters, but that some in some cases the higher standard of clear and convincing evidence is mandated.

See also Hope Brick Works v. Welch, 33 Ark. App. 103, 802 S.W. 2d 476, 478 (1991) . In a workers' compensation case, the court stated that Arkansas Stat. Ann. § 11-9-601 requires that a causal connection between the claimant's occupation or employment and the occupational disease from which he suffers be established by clear and convincing evidence.

(n77)Footnote 51. *See* King, *Causation, Valuation, and Chance In Personal Injury Torts Involving Preexisting Conditions and Future Consequences*, 90 Yale L. J. 1353 (1981).

(n78)Footnote 52. *See* King, *Causation, Valuation, and Chance in Personal Injury Torts Involving Preexisting Conditions and Future Consequences*, 90 Yale L. J. 1353 (1981).

(n79)Footnote 53. Hahn v. Weber & Sons Co., 223 Neb. 426, 390 N.W.2d 503, 506 (1986) .

(n80)Footnote 54. Manko v. United States, 636 F. Supp. 1419, 1435-1437 (D. Mo. 1986) , applying Missouri law.

(n81)Footnote 55. Meloni v. Johns-Manville Sales Corp., No. 81-2542, slip op. (D.N.J. Oct. 2, 1986), applying New Jersey law. The court based its decision on the plaintiff's failure to meet the Herber v. Johns-Manville Corp., 785 F.2d 79 (3d Cir. 1986) , standard that "plaintiff faces a risk of experiencing a harm which is *more likely than not* to occur.

(n82)Footnote 56. Mauro v. Raymark Industries, Inc., 116 N.J. 126, 561 A.2d 257, 1989 N.J. LEXIS 100, CCH Prod. Liab. Rep. P12234, 14 O.S.H. Cas. (BNA) 1161 . This case is also discussed in § 10.04[1][c].

(n83)Footnote 57. McClelland v. Goodyear Tire & Rubber Co., 735 F. Supp. 172, 173-174 (D. Md. 1990) .

(n84)Footnote 58. *See, e.g.,* Rizzo & Arnold, *Causal Apportionment in the Law of Torts: An Economic Theory,* 80 Colum. L. Rev. 1399 (1980) and Rosenberg, *above* note 1.

(n85)Footnote 59. Vuocolo v. Diamond Shamrock Chems. Co., 240 N.J. Super. 289, 573 A.2d 196, 201-202 (App. Div. 1990) .

(n86)Footnote 60. *See* Dannen & Sagill, note 14, *above*, at 304; *see also* Smith, *Admissibility of Expert Testimony and the Toxic Tort*, 15 J. Prod. and Tox. Liab. 97 (Spring 1993); Dreyer, *An Epidemiologic View of Causation: How It Differs From the Legal*, 61 Def. Couns. J. 40 (Jan. 1994).

(n87)Footnote 61. *See, e.g.,* Elam v. Alcolac, 765 S.W. 2d 42, 91-92 (Mo. App. 1988) .

(n88)Footnote 62. Allen v. United States, 588 F. Supp. 247, 1984 U.S. Dist. LEXIS 16822, 1984 U.S. Dist. LEXIS 16823 , applying Utah law.

(n89)Footnote 63. Allen v. United States, 588 F. Supp. 247, 1984 U.S. Dist. LEXIS 16822

(n90)Footnote 64. For a detailed discussion of statistical significance as it relates to the legal standard of proof, *see generally* Longmore v. Merrell Dow Pharmaceuticals, 737 F. Supp. 1117, 1119-1121 (D. Idaho 1990) .

(n91)Footnote 65. Hardcastle v. International Paper Co., No. 86-1110, slip op. (C.D. Ill. Feb. 4, 1988).

(n92)Footnote 66. Wheelahan v. G.D. Searle & Co., 814 F.2d 655 (4th Cir. 1987) .

(n93)Footnote 67. Washington v. Armstrong World Indus. Inc., 839 F.2d 1121, 1122-1123 (5th Cir. 1988) .

(n94)Footnote 68. Brock v. Merrell Dow Pharmaceuticals, Inc., 874 F.2d 307, 313-314 (5th Cir. 1989) , applying federal law. This case is also discussed in § 10.02[6][c].

(n95)Footnote 69. Davis & Rall, *Risk Assessment for Disease Prevention*, ALI-ABA Toxic Substances and Hazardous Wastes 623 (1980).

(n96)Footnote 70. **"Reasonable medical certainty" does not mean scientific certainty.** *See* Redmon v. Sooter, 274 N.E.2d 200, 204-205 (1971) ; Hope Brick Works v. Welch, 33 Ark. App. 103, 802 S.W.2d 476, 478 (1991) ; Rockwell Int'l v. Turnbull, 802 P.2d 1182, 1184 (Colo. Ct. App. 1990) ; Pippin v. St. Joe Minerals Corp., 799 S.W.2d 898, 906-908 (Mo. Ct. App. 1990) .

(n97)Footnote 71. Lasha v. Olin Corp., 625 So. 2d 1002, 1005 (La. 1993) .

(n98)Footnote 72. *See, e.g.,* Eggen, *Toxic Torts, Causation, and Scientific Evidence After Daubert*, 55 U. Pitt. L. Rev. 889 (Spring 1994); Slater, *Federal Standards For Admissibility of Expert Evidence on Causation*, 61 Def. Couns. J. 51 (Jan. 1994); Livingood, *Admissibility and Reliability of Expert Scientific Testimony After Daubert*, 61 Def. Couns. J. 19 (Jan. 1994); Note, *"Helpful" or "Reasonably Reliable?" Analyzing the Expert Witness's Methodology Under Federal Rules of Evidence 702 and 703*, 77 Cornell L. Rev. 350 (1992); Spyridon, *Scientific Evidence v. Junk Science*, 61 Miss. L.J. 287 (Fall 1991); Kaye, *Is Proof of Statistical Significance Relevant*, 61 Wash. L. Rev. 1333 (1986).

(n99)Footnote 73. Frye v. U.S., 54 App. D.C. 46, 293 F. 1013 (D.C. Cir. 1923) .

(n100)Footnote 74. Daubert v. Merrell Dow Pharms., Inc., 509 U.S. 579, 113 S. Ct. 2786, 125 L. Ed. 2d 469, 478 (1993).

(n101)Footnote 75. For a more detailed discussion of *Daubert, see* Eggen, *Toxic Torts, Causation, and Scientific Evidence After Daubert*, 55 U. Pitt. L. Rev. 889 (Spring 1994); Livingood, *Admissibility and Reliability of Expert Scientific Testimony After Daubert*, 61 Def. Couns. J. 19 (Jan. 1994); 105 A.L.R. Fed. 299.

(n102)Footnote 76. Evans, *Causation and Disease: The Henle-Koch Postulates Revisited*, 49 Yale J. Biol. & Med. 175-195 (1976).

(n103)Footnote 77. *See* Wheelahan v. G.D. Searle & Co., 814 F.2d 655 (4th Cir. 1987), applying federal and Maryland law (court upheld judgment for defendant on grounds that evidence that Copper 7 IUD "related to" or was associated with the type of injuries suffered by plaintiffs was inadequate scientific proof of causation).

(n104)Footnote 78. M. Wintrobe, Clinical Hematology 708 (Lea & Febiger, 8th ed. 1981).

On aplastic anemia, *see generally* 13 Courtroom Medicine, Ch. 8, *Cancer* (Matthew Bender).

(n105)Footnote 79. Evans, *Causation and Disease: The Henle-Koch Postulates Revisited,* 49 Yale J. Bio. & Med. 175-195 (1976).

(n106)Footnote 80. Evans, *Causation and Disease: The Henle-Koch Postulates Revisited,* 49 Yale J. Bio. & Med. 175, 189 (1976).

(n107)Footnote 81. Evans, *Causation and Disease: The Henle-Koch Postulates Revisited,* 49 Yale J. Bio. & Med. 175, 190 (1976).

(n108)Footnote 82. Lynch v. Merrell-National Laboratories, 646 F. Supp. 856, 864 (D. Mass. 1986), *aff'd,* 830 F.2d 1190, 1194 (1st Cir. 1987).

(n109)Footnote 83. Hackney & Linn, *Koch's Postulates Updated: A Potentially Useful Application to Laboratory Research and Policy Analysis in Environmental Toxicology,* 119 Am. Rev. Respir. Disease 849-852 (1979).

(n110)Footnote 84. Hackney & Linn, *Koch's Postulates Updated: A Potentially Useful Application to Laboratory Research and Policy Analysis in Environmental Toxicology,* 119 Am. Rev. Respir. Disease 849-850 (1979).

(n111)Footnote 85. Hackney & Linn, *Koch's Postulates Updated: A Potentially Useful Application to Laboratory Research and Policy Analysis in Environmental Toxicology,* 119 Am. Rev. Respir. Disease 849-850 (1979).

(n112)Footnote 86. Gots, *Medical Causation and Expert Testimony,* 96 Regulatory Toxic. & Pharmac. 98 (June 1986).

(n113)Footnote 87. Gots, *Medical Causation and Expert Testimony,* 96 Regulatory Toxic. & Pharmac. 98, 102 (June 1986).

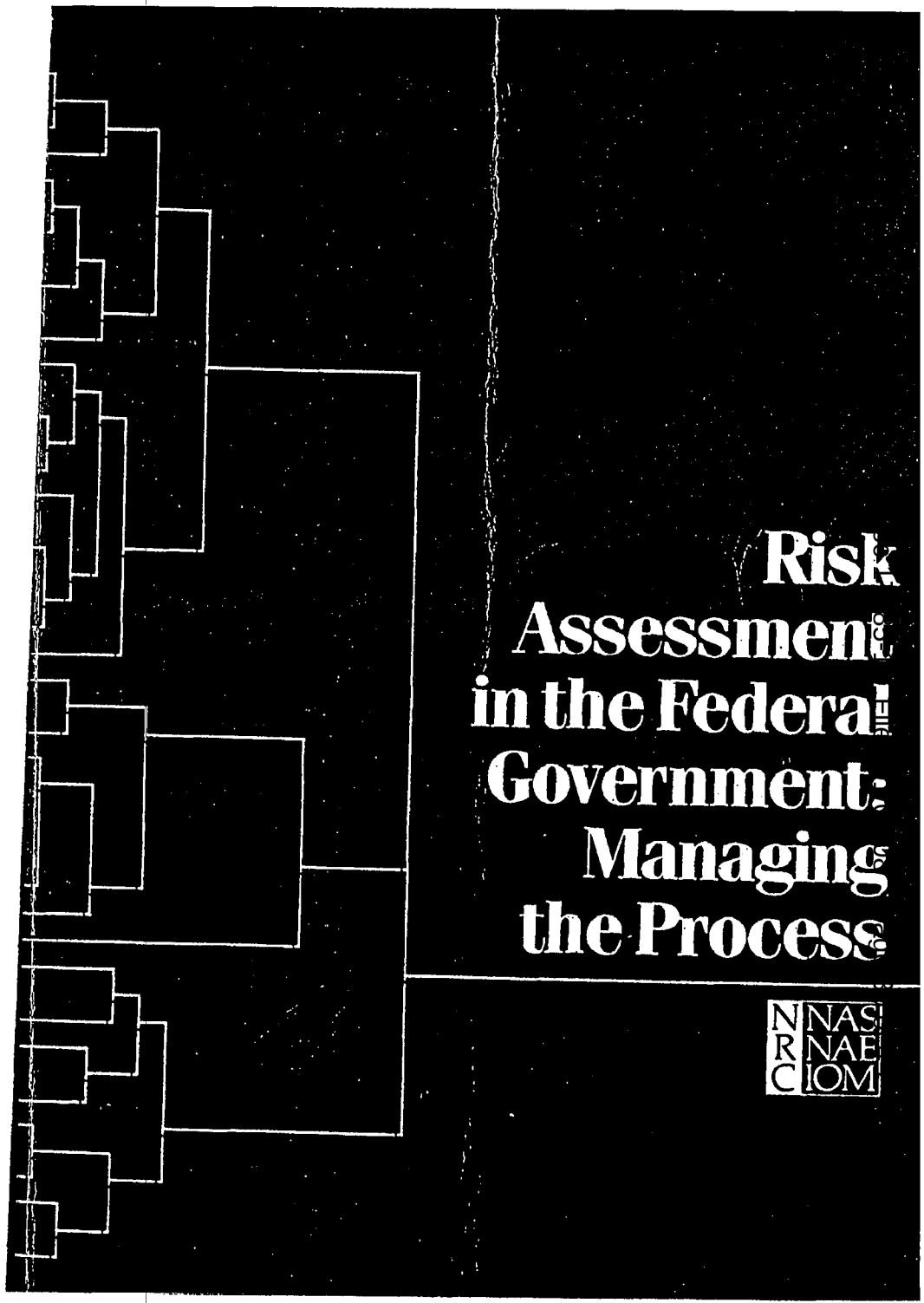
(n114)Footnote 88. *See* ch. 21, *How to Handle an Asbestos Case*; *see also* Courtroom Medicine, ch. 12B, *Chest, Heart and Lungs* (Matthew Bender).

(n115)Footnote 89. *See* Courtroom Medicine, ch. 9, *Cancer* (Matthew Bender).

* By Robert E. Shields, Esq., a partner in the Atlanta, Georgia law firm of Doffermyre, Shields, Canfield & Knowles.

Revised by Rachel Kerdman, Esq. Bayside, New York.

EXHIBIT 119


Risk
Assessment
in the Federal
Government:
Managing
the Process
NRC
NAS
NAE
IOM

Risk Assessment in the Federal Government: Managing the Process

Committee on the Institutional Means for Assessment of Risks to Public Health

Commission on Life Sciences

National Research Council

NATIONAL ACADEMY PRESS
Washington, D. C. 1983

NOTICE: The project that is the subject of this report was approved by the Governing Board of the National Research Council, whose members are drawn from the councils of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine. The members of the committee responsible for the report were chosen for their special competences and with regard for appropriate balance.

This report has been reviewed by a group other than the authors according to procedures approved by a Report Review Committee consisting of members of the National Academy of Sciences, the National Academy of Engineering, and the Institute of Medicine.

The National Research Council was established by the National Academy of Sciences in 1916 to associate the broad community of science and technology with the Academy's purposes of furthering knowledge and of advising the federal government. The Council operates in accordance with general policies determined by the Academy under the authority of its congressional charter of 1863, which establishes the Academy as a private, nonprofit, self-governing membership corporation. The Council has become the principal operating agency of both the National Academy of Sciences and the National Academy of Engineering in the conduct of their services to the government, the public, and the scientific and engineering communities. It is administered jointly by both Academies and the Institute of Medicine. The National Academy of Engineering and the Institute of Medicine were established in 1964 and 1970, respectively, under the charter of the National Academy of Sciences.

The study reported here was supported by Contract 223-81-8251 between the National Academy of Sciences and the Food and Drug Administration, Department of Health and Human Services.

Library of Congress Catalog Card Number 83-80381

International Standard Book Number 0-309-03349-7

Available from

NATIONAL ACADEMY PRESS
2101 Constitution Avenue, N.W.
Washington, D.C. 20418

Printed in the United States of America

First Printing, March 1983
Second Printing, October 1983
Third Printing, August 1984

NATIONAL RESEARCH COUNCIL

2101 CONSTITUTION AVENUE WASHINGTON, D. C. 20418

OFFICE OF THE CHAIRMAN

March 1, 1983

Arthur Hull Hayes, Jr., M.D.
Commissioner of Food and Drugs
Food and Drug Administration
5600 Fishers Lane
Rockville, MD 20857

Dear Dr. Hayes:

I am pleased to transmit the enclosed report entitled "Risk Assessment in the Federal Government: Managing the Process." This study was authorized by P.L. 96-528 and carried out by a committee of the National Research Council's Commission on Life Sciences with support from the Food and Drug Administration under Contract No. 223-81-8251.

The Congress made provision for this study to strengthen the reliability and objectivity of scientific assessment that forms the basis for federal regulatory policies applicable to carcinogens and other public health hazards. Federal agencies that perform risk assessments are often hard pressed to clearly and convincingly present the scientific basis for their regulatory decision. In the recent past, for example, decisions on saccharin, nitrites in food, formaldehyde use in home insulations, asbestos, air pollutants and a host of other substances have been called into question.

The report recommends no radical changes in the organizational arrangements for performing risk assessments. Rather, the committee finds that the basic problem in risk assessment is the incompleteness of data, a problem not remedied by changing the organizational arrangement for performance of the assessments. Instead, the committee has suggested a course of action to improve the process within the practical constraints that exist.

One proposal by the committee requires explanation. It would provide that there be established under Academy auspices a Board on Risk Assessment Methods. This recommendation emerges strictly from the committee's internal deliberation. The committee alone is responsible for the substantive contents and findings of the report. Were a request made to the Academy along the lines of that particular recommendation to establish such a Board, the request would be considered de novo by the appropriate governing bodies of the institution.

Yours sincerely,

Frank Press

Frank Press
Chairman

Committee on the Institutional Means for Assessment of Risks to Public Health

REUEL A. STALLONES, School of Public Health, University of Texas, Houston, Tex., Chairman
MORTON CORN, Department of Environmental Health Sciences, Johns Hopkins University School of Hygiene and Public Health, Baltimore, Md.
KENNY S. CRUMP, Science Research Systems, Inc., Ruston, La.
J. CLARENCE DAVIES, Conservation Foundation, Washington, D.C.
VINCENT P. DOLE, Rockefeller University, New York, N.Y.
TED R. I. GREENWOOD, Department of Political Science, Massachusetts Institute of Technology, Cambridge, Mass.
RICHARD A. MERRILL, University of Virginia School of Law, Charlottesville, Va.
FRANKLIN E. MIRER, Department of Health and Safety, International Union, UAW, Detroit, Mich.
D. WARNER NORTH, Decision Focus, Inc., Los Altos, Calif.
GILBERT S. OMENN, Department of Environmental Health, University of Washington School of Public Health and Community Medicine, Seattle, Wash.
JOSEPH V. RODRICKS, ENVIRON Corporation, Washington, D.C.
PAUL SLOVIC, Decision Research, A Branch of Perceptronics, Inc., Eugene, Oreg.
H. M. D. UTIDJIAN, American Cyanamid Company, Wayne, N.J.
ELIZABETH WEISBURGER, National Cancer Institute, National Institutes of Health, Bethesda, Md.

Staff

LAWRENCE E. McCRAY, Project Director
CATHERINE L. ST. HILAIRE, Staff Officer
WILLIAM M. STIGLIANI, Staff Officer
RENEE M. ST. PIERRE, Administrative Secretary
NORMAN GROSSBLATT, Editor

Acknowledgments

The Committee acknowledges with appreciation information provided by the following persons.

KARIM AHMED, Research Director, Natural Resources Defense Council

ELIZABETH ANDERSON, Director, Office of Health and Environmental Assessment, Environmental Protection Agency

EDWIN L. BEHRENS, Procter and Gamble Corporation (representing American Industrial Health Council)

JACKSON B. BROWNING, Union Carbide Corporation (representing Chemical Manufacturers Association)

WILLIAM D. CAREY, Executive Officer, American Association for the Advancement of Science

PETER F. CARPENTER, Vice President for Corporate Strategy, Alza Corp., Palo Alto, Calif. (representing Pharmaceutical Manufacturers Association)

PAUL F. DEISLER, JR., Vice President, Health Safety and Environment, Shell Oil Company

ROBERT I. FIELD, Research Associate, Analysis and Inference, Inc., Boston, Mass.

W. GARY FLAMM, Deputy Associate Commissioner for Health Affairs (Science), Food and Drug Administration

SHERWIN GARDNER, Vice President, Science and Technology, Grocery Manufacturers of America, Inc.

MICHAEL GOUGH, Health Program Project Director, Office of Technology Assessment, U.S. Congress.

THOMAS P. GRUMBLY, Senior Consultant, Temple, Barker and Sloan, Inc., Lexington, Mass.

PAUL T. HOPPER, General Foods Corporation (representing Social and Economic Committee, Food Safety Council)

NATHAN J. KARCH, Assistant Professor, Howard University; and Senior Science Advisor, Clement Associates

RAPHAEL G. KASPER, Executive Director, Commission on Physical Sciences, Mathematics, and Resources, NAS-NRC
ARNOLD M. KUZMACK, Chevy Chase, Md.
RICHARD LEMEN, Director, Division of Standards Development and Technology Transfer, National Institute of Occupational Safety and Health
JOHN MARTONIK, Deputy Director of Health Standards Programs, Occupational Safety and Health Administration
WILLIAM McCARVILLE, Monsanto Co. (representing American Industrial Health Council)
SANFORD A. MILLER, Director, Bureau of Foods, Food and Drug Administration
PAUL MILVY, Environmental Law Institute
WARREN R. MUIR, Visiting Associate Professor, Department of Environmental Sciences, Johns Hopkins University
DENIS PRAGER, Assistant Director for Life Sciences and Institutional Relations, Office of Science and Technology Policy, Executive Office of the President
PETER PREUSS, Associate Executive Director, Directorate of Health Sciences, Consumer Product Safety Commission
DAVID RALL, Director, National Institute of Environmental Health Sciences
WILLIAM D. ROWE, Director, Institute for Risk Analysis; and Professor of Decision and Risk Analysis, American University
JAMES H. SAMMONS, Executive Vice President, American Medical Association
SHELDON SAMUELS, Director, Health Safety and Environmental Industrial Union Department, AFL/CIO
BRUCE SILVERGLADE, Director for Legal Affairs, Center for Science in the Public Interest
M. J. SLOAN, Manager, Regulatory Affairs, Shell Oil Company
STEVEN M. SWANSON, Director, Health and Safety Regulation, American Petroleum Institute
ROBERT G. TARDIFF, Executive Director, Board on Toxicology and Environmental Health Hazards, NAS-NRC
MONTE C. THRODAHL, Monsanto Corporation (representing American Industrial Health Council)
HAROLD TRABOSH, Deputy Director, Residue Evaluation and Surveillance Division, Food Safety Inspection Service, Department of Agriculture
REP. WILLIAM C. WAMPLER (D-VA), U.S. House of Representatives

Preface

In response to a directive from the Congress of the United States, the Food and Drug Administration contracted with the National Academy of Sciences to conduct a study of the institutional means for risk assessment. The Committee on the Institutional Means for Assessment of Risks to Public Health was formed in the National Research Council's Commission on Life Sciences in October 1981 and completed its work in January 1983. The members of the Committee were chosen to represent a broad array of backgrounds and special skills, both in the technology of risk assessment and in the formulation and application of policy in this field, and brought together extensive experience in industry, government, and academic life.

The Committee, with outstanding staff support, reviewed much of the published literature on risk assessment, studied the structures and operations of federal regulatory and research agencies, analyzed the history of regulation of selected chemicals, and sought and received the judgments of some exceptionally knowledgeable people. We are most grateful for the assistance so generously provided to us, but, of course, the responsibility for this report is entirely ours.

The Committee has sought to examine and codify past experience with risk assessment and relate that experience to patterns and practices. Our judgments are necessarily subjective, but we have endeavored to be impartial. In the process, we developed a disinclination for sweeping changes; we believe that more gradual, evolutionary alterations will result in greater improvements in the conduct and use of risk assessment.

REUEL A. STALLONES
Chairman

Contents

Risk Assessment in the Federal Government: Managing the Process

Summary

SETTING

This report explores the intricate relations between science and policy in a field that is the subject of much debate--the assessment of the risk of cancer and other adverse health effects associated with exposure of humans to toxic substances. It is a report of a search for the institutional mechanisms that best foster a constructive partnership between science and government, mechanisms to ensure that government regulation rests on the best available scientific knowledge and to preserve the integrity of scientific data and judgments in the unavoidable collision of the contending interests that accompany most important regulatory decisions.

Many decisions of federal agencies in regulating chronic health hazards have been bitterly controversial. The roots of the controversy lie in improvements in scientific and technologic capability to detect potentially hazardous chemicals, in changes in public expectations and concerns about health protection, and in the fact that the costs and benefits of regulatory policies fall unequally on different groups within American society.

The decade of the 1970s was a period of heightened public concern about the effects of technology on the environment. Individuals and groups urged strict government regulation as scientific evidence emerged that various chemical substances may induce cancers or other chronic health effects in humans, and new government programs were established to control potential hazards. The evidence of health effects of a few chemicals, such as asbestos, has been clear; in many more cases the evidence is meager and indirect. To aid decision-making,

agencies have developed procedures for identifying chronic health hazards and estimating the risks to human health posed by products and activities. However, rather than alleviating the controversy attending regulatory decisions, the procedures themselves have become a focus of criticism by scientists, industry representatives, and public-interest groups.

STUDY OBJECTIVES AND SCOPE

The Committee on Institutional Means for Assessment of Risks to Public Health was formed, in response to a congressional directive, to fulfill three primary objectives:

- To assess the merits of separating the analytic functions of developing risk assessments from the regulatory functions of making policy decisions.
- To consider the feasibility of designating a single organization to do risk assessments for all regulatory agencies.
- To consider the feasibility of developing uniform risk assessment guidelines for use by all regulatory agencies.

The Committee considered the current practice of risk assessment and its relation to the process of regulation of hazards to human health, past efforts to develop and use risk assessment guidelines, the experience of government regulatory agencies with different administrative arrangements for risk assessment, and various proposals to modify risk assessment procedures. Our study was directed primarily, although not exclusively, to the issue of increased risk of cancer resulting from exposure to chemicals in the environment, an issue that has aroused great public concern in recent years, as illustrated by the controversies involving the control of saccharin, asbestos, and formaldehyde. Despite this emphasis, however, our conclusions and recommendations are applicable in some degree across the broad field of environmental health.

Criticisms of risk assessment have ranged broadly from details of the process to administrative management to statutory authority. The mandate to this Committee did not include examination of the scientific issues involved in risk assessment or the broad social policy questions

that have been raised. The Committee's more limited purpose was to examine whether altered institutional arrangements or procedures can improve regulatory performance.

THE NATURE OF RISK ASSESSMENT

Regulatory actions are based on two distinct elements, risk assessment, the subject of this study, and risk management. Risk assessment is the use of the factual base to define the health effects of exposure of individuals or populations to hazardous materials and situations. Risk management is the process of weighing policy alternatives and selecting the most appropriate regulatory action, integrating the results of risk assessment with engineering data and with social, economic, and political concerns to reach a decision.

Risk assessments contain some or all of the following four steps:

- Hazard identification: The determination of whether a particular chemical is or is not causally linked to particular health effects.
- Dose-response assessment: The determination of the relation between the magnitude of exposure and the probability of occurrence of the health effects in question.
- Exposure assessment: The determination of the extent of human exposure before or after application of regulatory controls.
- Risk characterization: The description of the nature and often the magnitude of human risk, including attendant uncertainty.

In each step, a number of decision points (components) occur where risk to human health can only be inferred from the available evidence. Both scientific judgments and policy choices may be involved in selecting from among possible inferential bridges, and we have used the term risk assessment policy to differentiate those judgments and choices from the broader social and economic policy issues that are inherent in risk management decisions. At least some of the controversy surrounding regulatory actions has resulted from a blurring of the distinction between risk assessment policy and risk management policy.

UNIFORM GUIDELINES FOR RISK ASSESSMENT

An inference guideline is an explicit statement of a predetermined choice among alternative methods (inference options) that might be used to infer human risk from data that are not fully adequate or are not drawn directly from human experience. For example, a guideline might specify the mathematical model to be used to estimate the effects of exposure at low doses on the basis of the effects of exposure at high doses.

Over the last 2 decades, most federal regulatory agencies and other institutions responsible for risk assessment of toxic chemicals have sought to develop such guidelines. Their efforts have met with varied success. Agencies have cited several reasons for writing guidelines: to provide a systematic way to meet statutory requirements, to inform the public and regulated industries of agency policies, to stimulate public comment on those policies, to avoid arguing generic questions anew in each specific case, and to foster consistency and continuity of approach. Interagency guidelines for carcinogens, although short-lived, were developed by the agencies of the Interagency Regulatory Liaison Group (IRLG) and adopted by the President's Regulatory Council in 1979. The stated objective of that effort was to reduce inconsistency, duplication of effort, and lack of coordination among the federal agencies.

The form of guidelines varies widely. Some guidelines are comprehensive and detailed, addressing most of the components of risk assessment and describing underlying scientific concepts; others address only a few broad principles. Guidelines differ greatly in their degree of flexibility, i.e., the degree to which they permit assessors to consider scientific evidence that may justify departures from the prescribed inference options. And they vary in the legal authority vested in them: some are adopted as formal regulations and others by less formal means.

The Committee concludes that guidelines are feasible and, if properly designed, desirable; that clear statements of the inferences to be made in each step would be of advantage to the regulatory agencies, to the industries concerned, and to the general public; and that guidelines should be used uniformly by the governmental agencies.

INSTITUTIONAL ARRANGEMENTS FOR RISK ASSESSMENT

Dissatisfaction with government regulatory actions has led to proposals to restructure the institutional arrangements for risk assessment by:

- Organizational separation of risk assessment from risk management.
- Centralization of risk assessment activities in a single organization to serve all the regulatory agencies.

Four federal agencies--the Environmental Protection Agency (EPA), Food and Drug Administration (FDA), Occupational Safety and Health Administration (OSHA), and Consumer Product Safety Commission (CPSC)--have been given primary authority to regulate activities and substances that pose chronic health risks, and these four agencies' past actions have inspired many of the proposals for institutional change. The Committee reviewed a number of agency structures and procedures in an attempt to determine the merits of institutional separation and centralization. Examples were selected to illustrate different degrees of separation and centralization in the four agencies. Independent scientific review panels have been used to obtain some of the advantages proposed for organizational separation, and some of their experiences were examined.

Cross-agency comparisons are difficult, because the regulatory agencies and their various programs differ markedly in structure, procedures, personnel characteristics, administrative history, and statutory direction. In addition, agencies and programs change, and practices adhered to for several years may be altered substantially. These practical limitations to the evaluation of agency structures and practices led the Committee to conclude that predicting the likely effects of organizational rearrangements on agency performance of risk assessment is unavoidably judgmental. However, the available evidence shows no clear advantage of one administrative structure over another.

CONCLUSIONS AND MAJOR RECOMMENDATIONS

Dissatisfaction with the actions of federal regulatory agencies is often expressed as criticism of the conduct and administration of the risk assessment process. The

Committee believes that the basic problem in risk assessment is the sparseness and uncertainty of the scientific knowledge of the health hazards addressed, and this problem has no ready solution. The field has been developing rapidly, and the greatest improvements in risk assessment result from the acquisition of more and better data, which decreases the need to rely on inference and informed judgment to bridge gaps in knowledge.

Proposals to separate the administrative responsibility for risk assessment from risk management imply that the change would lead to improved risk assessment and hence better risk management decisions. Administrative relocation will not, however, improve the knowledge base, and, because risk assessment is only one element in the formulation of regulatory actions, even considerable improvements in risk assessment cannot be expected to eliminate controversy over those actions.

Organizational separation may have the advantage of establishing firmly the distinction between risk assessment and risk management, but it also has some disadvantages. The importance of distinguishing between risk assessment and risk management does not imply that they should be isolated from each other; in practice they interact, and communication in both directions is desirable and should not be disrupted. Institutional separation would surely reduce the responsiveness of the risk assessment process to the needs of the regulatory agencies for timely reports in accord with their priorities. In addition to the operational disadvantages, the disruption of current patterns of activity would be great, and the benefits uncertain. On balance, the Committee believes that transfer of risk assessment functions to an organization separate from the regulatory agencies is not appropriate.

We believe that risk assessment can be improved more surely and more effectively by adopting a program with three major parts: (A) implementation of procedural changes to ensure that individual assessments routinely take full advantage of the available scientific knowledge, while preserving the diversified approaches to the administration of risk assessment necessary to accommodate the varied needs of federal regulatory programs; (B) standardization of analytic procedures among federal programs through the development and use of uniform inference guidelines; and (C) creation of a mechanism that will ensure orderly and continuing review and modification of

risk assessment procedures as the scientific knowledge base expands.

(A) We recommend that regulatory agencies take steps to establish and maintain a clear conceptual distinction between assessment of risks and consideration of risk management alternatives; that is, the scientific findings and policy judgments embodied in risk assessments should be explicitly distinguished from the political, economic, and technical considerations that influence the design and choice of regulatory strategies.

We agree with proponents of such measures as the American Industrial Health Council's proposed science panel and H.R. 638 that efforts should be made by regulators and others to distinguish clearly between the assessment of risk and the choice of regulatory options.

We advocate the adoption of specific procedural measures that can be introduced under current arrangements. These measures include timely independent scientific review of major agency risk assessments and, to facilitate both scientific and public review of risk assessments, the routine preparation of written risk assessments that explicitly state the basis of choice among inference options.

(B) We recommend that uniform inference guidelines be developed for the use of federal regulatory agencies in the risk assessment process.

The Committee endorses the development and use of guidelines for risk assessment. These guidelines, which would structure the interpretation of scientific and technical information relevant to the assessment of health risks, should be followed by all federal agencies. They should address all elements of risk assessment, but allow flexibility to consider unique scientific evidence in particular instances.

The use of uniform guidelines would promote clarity, completeness, and consistency in risk assessment; would clarify the relative roles of scientific and other factors in risk assessment policy; would help to ensure that assessments reflect the latest scientific understanding; and would enable regulated parties to anticipate government decisions. In addition, adherence to inference

guidelines will aid in maintaining the distinction between risk assessment and risk management.

(C) We recommend to the Congress that a Board on Risk Assessment Methods be established to perform the following functions:

(1) To assess critically the evolving scientific basis of risk assessment and to make explicit the underlying assumptions and policy ramifications of the inference options in each component of the risk assessment process.

(2) To draft and periodically to revise recommended inference guidelines for risk assessment for adoption and use by federal regulatory agencies.

(3) To study agency experience with risk assessment and evaluate the usefulness of the guidelines.

(4) To identify research needs in the risk assessment field and in relevant underlying disciplines.

The Committee concludes that success in improving the risk assessment process requires the establishment of an independent board of scientific stature. Such a board can serve as a continuing locus of discussion about ways to improve scientific and procedural aspects of risk assessment.

Introduction

Through Congress the American public has granted authority to federal administrative agencies to restrict private actions, such as the production and use of chemicals, when this is deemed necessary to protect the health of the public. The 1970s are notable for the large number of new federal regulatory laws that are applicable to the environment, both in the workplace and in the community. These laws reflect a dramatic and relatively rapid shift in public priorities toward the protection of health. Concurrently with shifts in social priorities, advances in science have contributed to policy problems, for the advances have revealed the extent of the environmental health problem. Some earlier regulatory programs had addressed exposure to toxic chemicals, but they were directed mainly at the risk of poisoning and other acute effects. Much policy-making related to such effects involved routine, short-term, acute animal studies to establish "no-observed-effect" doses and then the straightforward calculation of allowable human exposure based on the application of safety factors to relatively uncomplicated scientific findings. Such an approach reflected little recognition of problems that might be associated with smaller exposures. Cancer, birth defects, and other conditions were seldom seen as preventable by government intervention. Only in the last 15 years has the potential extent of the linkage between such conditions and toxic substances been revealed. The often-cited estimate that a large fraction of all cancers may be attributed to human exposure to toxic agents (including smoking, diet, lifestyle, and occupation) originated fairly recently (Boyland, 1969; Higginson, 1969), and it

was not until the 1970s that regulatory agencies focused their attention on cancer and other chronic health risks.

Scientific advances entered the picture in a second way. The technology that has made it possible to detect relations between particular agents and cancer or other chronic effects has evolved rapidly from the days when exposure through skin-painting and subcutaneous injection were relied on in animal tests of carcinogenicity. Increasingly, epidemiologic investigations have either confirmed the findings of animal experiments or provided evidence that linked exposures to particular chemicals to particular chronic health effects. The introduction of reliable testing methods resulted in broader government testing requirements and, steadily, the discovery of more and more suspect chemicals--many of them in common use--that demanded agency attention. The techniques are still developing, and we are still looking for better ways to design and interpret animal bioassay experiments.

The increase in newly suspect chemicals was accompanied by the development of instruments and procedures that permitted the detection of chemicals at lower and lower concentrations. Even if the number of suspect chemicals had not increased dramatically, these sensitive detection methods would have revealed the presence of such chemicals in concentrations that earlier methods would have missed. Combined with all those changes were the development and refinement of analytic methods of estimating the degree of human risk on the basis of data from human studies and animal experiments.

Public policies are not immediately adaptable to rapid changes in social priorities and scientific advances. Many of the fundamental difficulties of regulatory risk assessment result from attempts to bend old laws and policies to fit newly perceived risks. For instance:

- A regulatory framework based on the traditional approach involving no-observed-effect doses and safety factors is now being applied to health effects for which a no-effect dose cannot be demonstrated, except at zero exposure.
- Regulatory laws and programs designed for the elimination of what was understood to be the very rare event of chronic hazard now operate in the presence of the recognition that many agents are suspect.
- Agencies must evaluate hundreds of chemicals on which no data related to human risk are available and on

which few animal tests were required and many other chemicals that were tested with methods that do not meet modern standards.

- Laws were written and programs designed before current quantitative methods for estimating human risks on the basis of data from animal studies were developed.

DIFFICULTIES IN DECISION-MAKING

Agency decisions regarding potential carcinogens and similar hazards are commonly beset by two types of difficulties: inherent limitations on the power of analysis and practical constraints imposed by external pressures. Several such factors are particularly relevant to the consideration of scientific aspects of risk assessment.

INHERENT LIMITATIONS

Uncertainty

The dominant analytic difficulty is pervasive uncertainty. Risk assessment draws extensively on science, and a strong scientific basis has developed for linking exposure to chemicals to chronic health effects. However, data may be incomplete, and there is often great uncertainty in estimates of the types, probability, and magnitude of health effects associated with a chemical agent, of the economic effects of a proposed regulatory action, and of the extent of current and possible future human exposures. These problems have no immediate solutions, given the many gaps in our understanding of the causal mechanisms of carcinogenesis and other health effects and in our ability to ascertain the nature or extent of the effects associated with specific exposures. Because our knowledge is limited, conclusive direct evidence of a threat to human health is rare. Fewer than 30 agents are definitely linked with cancer in humans (Tomatis *et al.*, 1978); in contrast, some 1,500 substances are reportedly carcinogenic in animal tests, although they include substances tested in studies of questionable experimental design. We know even less about most chemicals; only about 7,000 of the over 5,000,000 known substances have ever been tested for carcinogenicity (Maugh, 1978)--a small fraction of those theoretically under regulatory jurisdiction. We

know still less about chronic health effects other than cancer.

Ethical considerations prevent deliberate human experimentation with potentially dangerous chemicals, and the length of the latent period for cancer and some other effects greatly complicates epidemiologic studies of uncontrolled human exposures. Animal models must be used to investigate whether exposure to a chemical is related to the incidence of health effects, and the results must be extrapolated to humans. To make judgments amid such uncertainty, risk assessors must rely on a series of assumptions.

Limited Analytic Resources

The number of chemicals in the jurisdiction of federal regulatory agencies is enormous. For example, of the roughly 5,000,000 known chemicals, more than 70,000 are in commercial use (Fishbein, 1980). The Environmental Protection Agency's Chemical Activities Status Report lists about 3,500 chemicals as being under some sort of active consideration in the Agency's various regulatory programs. Similarly, the Food and Drug Administration's food program must cope with over 2,000 food-related chemicals (900 flavors, 700 items listed as "generally recognized as safe," 350 food additives, 175 animal drugs, and 60 color additives) and an additional 12,000 indirect additives (Flamm, 1981).

The many problem chemicals in an agency's jurisdiction compete for attention of analysts and decision-makers. If an agency is considering new action on many substances at once, its scientific staff is stretched thin. Most agencies do not have the analytic resources to do a thorough risk assessment for priority-setting and must rely on less formal methods to ensure that the highest-risk chemicals are examined first.

Complexity

For most chemical agents that might be subject to regulation, a great variety of factors must be assessed, including potential toxicity, extent of human exposure, effectiveness of technologies to reduce exposure, the nature of possible substitute chemicals, effects on and interests of various population groups, and economic effects of

regulatory alternatives. Decision-makers in a regulatory agency may encounter a large amount of highly technical information as they work toward their decisions; many scientific disciplines and technical fields are usually involved. An agency would like to have simple rules and analytic procedures to ensure consistency and competence in its decision-making, but, in the face of scientific uncertainty, such simplicity is difficult to achieve without an inadvertent loss of crucial scientific insight from the decision process.

EXTERNAL PRESSURES

Public Concern with Health Protection

When the risk involves a serious disease, such as cancer, or birth defects, feelings are likely to run high, particularly if the groups exposed to a chemical are mobilized to express themselves in an agency's deliberations. Such groups insist that regulatory action need not await conclusive evidence of cause and effect and need not be based exclusively on the most scientifically advanced testing methods.

Visible Economic Interests

Although it is rarely known which individuals are likely to be saved from adverse health effects through a regulation that reduces exposure to a particular chemical, those who bear the economic costs of such restrictions can identify themselves without any difficulty. These parties can provide relatively concrete projections of a prospective regulation's inflationary influence, effect on employment, and other immediate economic effects, and such consequences may be substantial. They may question the wisdom of balancing concrete evidence of economic damage against evidence of health protection that depends on a complex series of assumptions derived from sparse and indirect data.

Congressional Action

In fulfilling its role as the legislative voice of popular concerns, Congress can act in ways that influence decision

processes. It can dictate the factors to be included in and excluded from decision-making (the Delaney clause is an example), and it can pass special legislation to preempt agency discretion, as it did in acting to prevent the removal of saccharin from the market.

PROPOSED REFORMS

Under these conditions, it would perhaps be surprising if calls for major reform were not heard. Some have sought to improve the techniques that the government uses to analyze and evaluate risks; for example, the House of Representatives in 1982 passed H.R. 6159 (commonly known as the "Ritter bill"), to establish a government-wide program of research and demonstration projects on quantitative and comparative risk analysis.

Much of the recent controversy is general; it reflects the conflict in values between different groups in society, particularly with regard to the relative importance of economic factors and health protection in the formulation of regulatory decisions. Different groups will inevitably disagree about the degree of risk (if any) that is defined as acceptable in a particular case. However, some criticisms directly address the risk assessment component of the overall decision-making process. Some critics question whether current practices adequately safeguard the quality of the scientific interpretations needed for risk assessment. With a scientific base that is still evolving, with large uncertainties to be addressed in each decision, and with the presence of great external pressures, some see a danger that the scientific interpretations in risk assessments will be distorted by policy considerations, and they seek new institutional safeguards against such distortion.

Among the institutional reforms suggested, two major categories are the focus of this report: reorganization to ensure that risk assessments are protected from inappropriate policy influences and development and use of uniform guidelines for carrying out risk assessments.

Some argue that scientific quality, consistency, and distinction between scientific judgment and policy judgment can be improved through the use of explicit guidelines for agency risk assessments. Such guidelines would specify methods for interpreting scientific data and would seek to limit analysts who confront data gaps or

extrapolation questions to methods that are consistent with the best current scientific judgment. Analysts following the guidelines would find it easier to describe systematically and explicitly the methods that are incorporated in their risk assessments.

Several other recent proposals call for major restructuring of federal processes to separate the risk assessment function organizationally from decision-making. The objectives would be to permit analysts to work independently of policy pressures and to foster consistency of risk assessments. Various approaches have been suggested, including creation of a single body outside the government for the performance or review of risk assessments, creation of a single government unit to conduct risk assessments for the entire government, and creation of separate risk assessment units in particular programs or agencies and systematic review of assessments by independent scientific advisory groups.

THE STUDY

This report responds to a congressional request to examine the merits of the two major types of reform proposal. It is the final report of the National Research Council's Committee on the Institutional Means for Assessment of Risks to Public Health. Chapter I describes the structure of risk assessment, the role of science in the assessment process, and current federal uses of risk assessment. Chapter II examines the feasibility and desirability of the development and use of uniform guidelines. Chapter III reviews various organizational arrangements for risk assessment. The Committee's overall conclusions and recommendations appear in Chapter IV.

REFERENCES

Boyland, E. 1969. The correlation of experimental carcinogenesis and cancer in man. Prog. Exp. Tumor Res. 77:222-234.

Fishbein, L. 1980. Potential industrial carcinogenic and mutagenic alkylating agents, pp. 329-363. In D. B. Walters, ed. Safe Handling of Chemical Carcinogens, Mutagens, Teratogens, and Highly Toxic Substances, vol. I. Ann Arbor, Mich.: Ann Arbor Science.